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VOL. I.—16TH YEAR. SYDNEY, SATURDAY, MAY 18, 1929. No. 20.

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THE INTRAVENOUS INJECTION OF PHOSPHATES: AN EXPERIMENTAL STUDY WITH REGARD TO DIAGNOSTIC AND THERAPEUTIC POSSIBILITIES IN CHRONIC INTERSTITIAL NEPHRITIS.[1]

By ADOLPH BOLLIGER, Ph.D.,
Director of Research, Department of Urology, University of Sydney.

THE production of a chronic interstitial nephritis in dogs by means of X radiation has been previously described.[(1)] Diagnostic and therapeutic studies in this experimental disease were attempted and some of the results with the usual renal function tests[(2)] as well as with the sodium thiosulphate and sodium iodide test[(3)] have been reported.

The present investigation deals with the excretion of sodium phosphate which was administered intravenously both to normal dogs and to nephritic dogs throughout the course of the disease. The diagnostic aspects of the excretion of sodium phosphate, together with some of the pharmacological and pathological features have been studied and are discussed in this paper.

Technique.

Sodium phosphate solutions of varying hydrogen ion concentration (pH 4·5 to pH 8·0), containing from 90 to 470 milligrammes of phosphorus in twenty cubic centimetres of water, were made up. After one night's fasting female dogs were injected with twenty cubic centimetres of one of the phosphate solutions, the bladder being previously emptied by catheterization.

Two hours after injection the dogs were again catheterized and the bladder irrigated with water. The phosphorus in the two-hour urine sample and the wash water was determined by the method of Benedict and Theis.[(4)] The methods used for chemical examination of the blood were those described in the previous papers.

The two-hour phosphate excretion test described above (called in future phosphate test) was applied (i) to normal dogs and dogs with extrarenal lesions and (ii) to dogs with different degrees of renal impairment produced by X rays.

Table I represents the findings in a normal dog of nineteen kilograms body weight, which was subjected to fifty-four phosphate injections over a period of seven months. On twenty-one occasions the two-hour phosphate output was determined. The phosphate solutions injected varied in phosphorus content and in hydrogen ion concentration in order to determine the influence of variations in these two factors on the normal excretion.

In this experiment the urea nitrogen content of the blood was frequently determined, but was never found above the normal limits, that is fifteen milligrammes per hundred cubic centimetres. The carbon dioxide combining power of the plasma also remained between normal limits (39% to 45%). The inorganic phosphate of the plasma rose immediately after injection, but had fallen to less than 4·5 milligrammes per hundred cubic centimetres (the normal figure) in three hours.

[1] From the research laboratory of the Department of Urology, University of Sydney, N.S.W. Most of the experimental observations described in this paper were made at the laboratory of the Henry Ford Hospital, Detroit, U.S.A.

TABLE I.
Normal Dog.

Date.	Phosphorus Excretion.	pH Values.	Milligrammes of Phosphorus Injected.
	%		
July 28, 1927	93	7·1	430
August 12, 1927	117	7·4	90
August 15, 1927	85	7·1	430
August 24, 1927	94	7·2	480
August 31, 1927	100	7·4	240
September 10, 1927	93	7·5	350
October 10, 1927	99	7·6	390
October 18, 1927	86	7·3	370
October 22, 1927	96	7·5	420
November 5, 1927	97	7·3	430
November 30, 1927	95	7·3	430
January 7, 1928	99	8·0	430
January 9, 1928	91	7·4	430
January 11, 1928	92	7·9	470
January 13, 1928	95	7·9	470
January 18, 1928	84	7·9	470
January 25, 1928	80	7·1	430
February 3, 1928	98	8·0	420
February 9, 1928	94	4·5	420
February 11, 1928	89	4·5	420
February 25, 1928	82	8·0	420

During the entire experiment the dog showed no clinical signs of illness, except that the urine frequently contained moderate amounts of albumin.

The animal was killed two months after the last injection. At autopsy all the organs, including the liver and kidneys, were of normal macroscopic appearance; no microscopic studies of the organs were made.

TABLE II.

Dog.	Phosphorus Secretion.			Remarks.
	First Test.	Second Test.	Third Test.	
	%	%	%	
1	95	101	98	Severe diabetic, depancreatized.
2	93	87	96	Complete heart block.
3	82	79	84	Extensive X ray burn.
4	86	82	92	Normal.
5	81	80	87	Normal.
6	81	86	89	Normal.
7	89	92	84	Normal.

Table II shows the results of three phosphate tests on seven more dogs. A number of these dogs are normal subjects, included for the purpose of a further demonstration of the normal limits of the test. The remainder had extrarenal lesions and were investigated to ascertain whether or not the phosphate test might be regarded as a specific renal test. The animals in Table II had from eight to twenty-five phosphate injections. No untoward effects were noted with the exception of occasional albuminuria.

Nephritic Dogs.

In this group six dogs with renal insufficiency of widely varying degree were given phosphate injections.

TABLE III.

Date.	Phosphorus Excretion. %	Phenol-sulphone-phthalein. %	Blood Urea N. Milligrammes %	Blood Inorganic Phosphorus. Milligrammes %
October 10, 1927	82	70	14	4·0
October 11, 1927	*a*			
October 21, 1927	*b*			
October 27, 1927	62	58	72	4·6
November 7, 1927	29	20	59	4·1
November 9, 1927	34	22	82	4·8
November 26, 1927	22	—	72	5·6
December 2, 1927	21	16	78	8·9
December 8, 1927	Trace	0	184	16·0
December 9, 1927	Died	—	—	—

a = left ureter tied. *b* = irradiation of the right kidney

Table III gives the findings in a dog with a very rapidly progressing nephritis complicated by a terminal pneumonia. In a preliminary operation the left ureter of this animal was ligated. Fourteen days later the right kidney was surgically exposed and rayed directly with a heavy dose (dosage 1,100 milliampère minutes, fifty centimetres kidney distance, filter 0·25 millimetres of copper).

The phosphate solutions injected ranged from pH 7·2 to pH 7·4. The rapid rise in blood urea content and the phosphate retention, together with the low phenolsulphonephthalein and phosphate excretion, prove the rapid failing of the right kidney.

Autopsy revealed a large uninfected hydronephrosis on the left side, following the complete obstruction of the left ureter. The right kidney was contracted to half the normal size and was pale and fibrous. In microscopical appearance it was of the subacute chronic type, as previously described in rapidly progressive nephritis due to X ray exposure.

The lungs showed gross signs of acute terminal pneumonia.

Chart I illustrates a case in which a greater number of phosphate injections was given and the advanced nephritis of the animal remained stationary for six months. The kidney insufficiency was produced by direct irradiation of the left kidney, the right kidney having been removed one month previously. The twenty-five phosphate injections given were of slightly higher hydrogen ion concentration than that of the blood (pH 7·1 to pH 7·2).

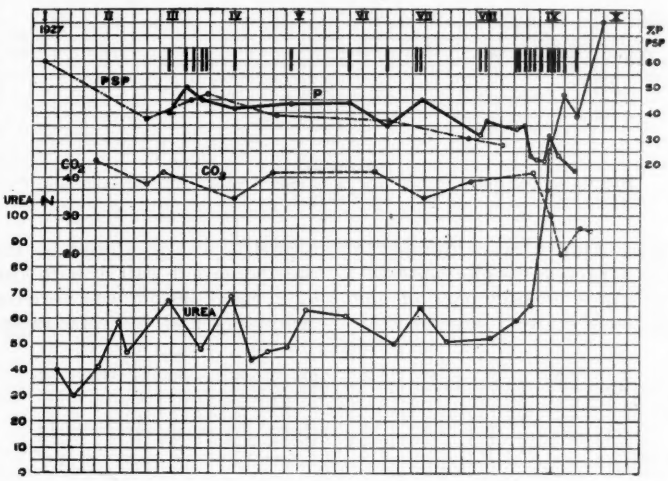

CHART I.

Results of Phosphate Injections in Nephritic Dog. Heavy vertical lines indicate phosphate injections. Heavy line curve indicates percentage of phosphates excreted. PSP indicates phenolsulphonephthalein excretion (—·—·—·). CO_2 indicates carbon dioxide combining power of the plasma (— — — —). The urea nitrogen content of the blood is indicated by a light continuous curve.

Throughout the six months of the experimental period the animal remained in an excellent state of nutrition and physical health until the last week of its life. In spite of a great degree of nephritis not as much vomiting occurred as in similar untreated animals. The dog had a sudden breakdown and died in a few days.

The only pathological condition revealed at autopsy was a contracted, pale kidney with a large calculus, filling the entire pelvis. The stone appeared to have recently obstructed the flow of urine into the ureter to a considerable extent and was responsible for the animal's sudden breakdown.

Microscopical examination of the kidney showed peculiar salt deposits in the basement membranes of the walls of the remaining tubules and in the capsule of Bowman over large areas (see Figure I). Figure II shows small salt deposits in a fibrosed area and in the capsular space of an atrophic glomerulus.

Chart II gives the findings in a fair-sized Airedale terrier whose right ureter was tied three weeks after irradiation of the left kidney. This dog received fifty-five phosphate injections over a period of six and a half months. The solutions administered contained from 380 to 450 milligrammes of phosphorus and were slightly more alkaline than the blood (pH 7·6 to 8·0). A rather severe nephritis was already present when the injections were started. The urea nitrogen was 63 milligrammes per hundred cubic centimetres, the phenolsulphonephthalein output 46%. The animal was vomiting occasionally and was in an undernourished condition.

About one month after the administration of phosphates the animal improved physically. The body weight rose from 14·0 to 14·8 kilograms and remained constant through the following month in spite of acidosis and severe urea retention.

Frequent determination showed that the calcium content of the serum remained at a raised level throughout the experiment. The figures varied from 11·8 to 13·4 milligrammes per hundred cubic centimetres, averaging 12·6 milligrammes. The inorganic phosphate determinations were made the day following injection. For four months no increased values were found. Two months before

death the phosphates were above five milligrammes per hundred cubic centimetres and values above eight milligrammes were noticed two weeks prior to death. As rupture of the huge hydronephrosis on the right side was feared, a right nephrectomy was done six months after the phosphate injections were started. After the operation the animal began to lose weight. Phosphate injections were given repeatedly. Four weeks after the operation the animal passed bloody stools. A few days later the dog vomited two hundred cubic centimetres of pure blood. As the hæmorrhage could not be stopped by means of blood transfusion, the dog was killed.

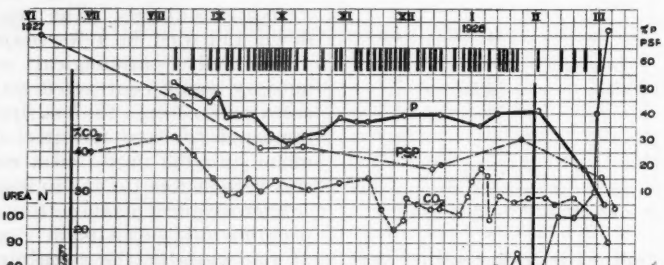

CHART II.
Results of Phosphate Injections in Nephritic Dog. For explanation see Chart I.

At autopsy three-fourths of the remaining kidney were found to consist of light yellow, contracted, fibrous material. The microscopical appearance of this part of the gland was found, as is usual in very advanced X ray nephritis, with the exception of some areas showing the same peculiar calcifications of the basement membranes of the tubular walls, as well as of the capsule of Bowman. These changes were demonstrated in the previous animal (see Figure III).

The upper pole of the kidney, constituting about one-quarter of the organ, was of normal macroscopical appearance. Figure IV illustrates the microscopical structure of a transitional area between the almost completely fibrosed area and the apparently normal pole. No tubules can be made out in the fibrosed area. The glomeruli are atrophic and two large salt deposits can be seen. On the left side of the picture the apparently normal pole, the only kidney substance which has been functioning, can be seen. Numerous tubules can be made out. Many have lost their epithelial lining and are dilated, while the majority of tubules has a fairly normal appearance.

The entire intestinal tract presented the picture of a severe hæmorrhagic enteritis, most evident in the stomach wall. Figure V depicts the microscopical appearance of part of the stomach wall. Vegetations were present on the pulmonary valves of the heart.

Chart III gives the findings in a dog belonging to a group of three animals. This dog had a less severe and more slowly progressing renal lesion than the three animals described above. The nephritis was produced by the irradiation through the abdominal wall of both kidneys. The production of a renal lesion was revealed by typical urinary symptoms and by constantly raised urea content of the blood. The phenolsulphonephthalein output varied between 55% and 60%. The first determination of phosphate excretion yielded only 60%.

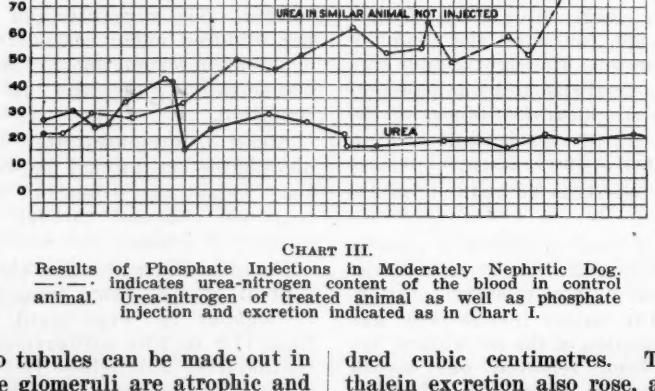

CHART III.
Results of Phosphate Injections in Moderately Nephritic Dog. —·—·— indicates urea-nitrogen content of the blood in control animal. Urea-nitrogen of treated animal as well as phosphate injection and excretion indicated as in Chart I.

In the course of the experiment phosphate solutions (pH 7·2) were injected less frequently than in previous experiments. In this case the phosphate output rose to normal limits after about seven injections. The urea nitrogen content fell to a lower level than before the phosphate injections and remained stationary at about twenty milligrammes per hundred cubic centimetres. The phenolsulphonephthalein excretion also rose, averaging 65%.

The animal improved physically. During the course of injections the body weight rose from

fifteen to sixteen kilograms. After seven months the experiment had to be interrupted, as the dog suddenly showed signs of severe pulmonary infection and had to be killed.

It was possible to carry out a control to this experiment; a similar dog of the same breed and age received the same X ray treatment and was left under similar conditions with the exception that it had no phosphate injections. For comparison the blood urea figures of the control animal have been included in Chart III.

At autopsy the phosphate-injected dog showed the ordinary picture of acute canine pneumonia. The only remarkable organs were the kidneys. In Figure VI the condition of the right kidney which is contracted to an extreme degree, weighing only sixteen grammes, is seen. The tissue around the hilum has a whitish appearance and is completely fibrosed. A small layer of kidney tissue having a normal appearance may be seen at the upper and lower poles. On microscopical examination normal and moderately fibrosed areas of cortex can be made out in this part.

The left kidney, weighing fifty-five grammes, also appears to be rather small. Except for a whitish area of complete fibrosis at the lower pole, four millimetres in length, the organ is of normal colour and consistency. On microscopical examination this organ shows healthy structure throughout with the exception of a few small and scattered fibrous patches. No damage was found which could be specifically attributed to the phosphate administration.

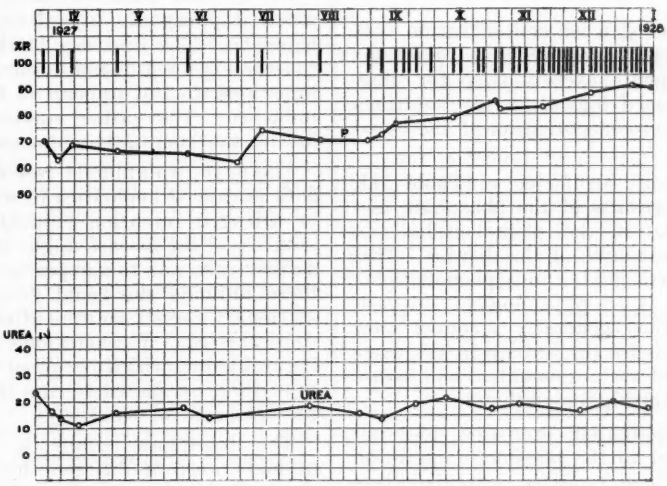

CHART IV.
Results of Phosphate Injections in Moderately Nephritic Dog. Phosphate injection and excretion as well as urea-nitrogen of the blood indicated as in Chart I.

Chart IV represents the findings in an animal which had the left kidney removed in July, 1926. During the following four months the right kidney was exposed to X rays through the abdominal wall for fifteen minutes on five occasions. The technique was the same as the one described in a previous paper.[1]

In March, 1927, before phosphate injections were started, a moderate and so far slowly progressing nephritis was found to be present. Urea nitrogen content of the blood ranged from twenty to forty milligrammes per hundred cubic centimetres. A constant, severe albuminuria with frequent hæmaturia was present (200 to 800 milligrammes of albumin per hundred cubic centimetres of urine). The animal was thin, body weight about fifteen kilograms, and vomited about twice a week.

After the first six injections the two hour phosphate output ranged from 63% to 70%.

After these first series of injections it became evident that the phosphate excretion was slowly rising. During the seventh month of the experiment the normal excretion of 80% or more was reached. Still later the results of the phosphate test were between 85% and 93%.

Coincident with the gradual rise in phosphate excretion the general health of the animal improved. Vomiting ceased and the body weight rose to sixteen kilograms.

As many as thirteen injections per month were given in the later part of the experiment. In spite of the greatly increased number of injections the animal remained in remarkably good health. The experiment was discontinued because the dog became pregnant. The pregnancy took a normal course.

The last animal in this group was subjected to X rays over both kidneys through the abdominal wall. It showed a similar course to the one described in Chart IV. Forty-eight injections were given over a period of eleven months. During this period the renal disease did not advance as expected, but rather improved slightly. The general health also improved and the phosphate excretion rose to normal levels. The urea nitrogen decreased from an average of thirty milligrammes to an average of twenty milligrammes per hundred cubic centimetres. The phenolsulphonephthalein excretion showed an average increase of about 5%.

Finally the animal was killed. The effect of the X rays on the kidney is shown by well localized scars in both kidneys, measuring about one centimetre in diameter. Except for these scars the kidneys show a normal structure, although they are reduced in size.

Comment.

Wigglesworth and Woodrow[5] have recently investigated the relation between the phosphate of the blood and those of the urine after the ingestion of phosphates by mouth. Addis, Meyers and Bayer[6] gave phosphates intravenously to rabbits and studied the effect of the variation of plasma phosphate concentration upon the rate of phosphate

excretion. As far as is known no attempt has yet been made to use the urinary excretion of sodium phosphates, administered intravenously, as a two-hour renal function test.

A normal dog, taken after a fairly short fasting period, will excrete from 80% to 100% of a solution of phosphate in two hours, when the phosphate solution has the properties described above and is given intravenously. Dogs with extrarenal lesions excrete the same amount (see Tables I and II).

Phosphates are always present in normal urine, but may vary greatly in amount. Consequently rather large doses had to be injected to produce reliable results. Three hundred and fifty milligrammes are sufficient to obtain constant findings, but for reasons to be discussed later it was usually my practice to give larger amounts (400 to 470 milligrammes).

Variations in the hydrogen ion concentration (pH 4·5 to pH 8·0) produced no appreciable variations in the two-hour phosphate excretion, so for diagnostic purposes it seemed logical to use solutions having the hydrogen ion concentration of the blood (pH 7·3 to 7·4).

In animals with renal insufficiency (Table III, Charts I and II) the results of the phosphate test run parallel to those of the phenolsulphonephthalein test and to the course of the disease. This agreement between phosphate and phenolsulphonephthalein excretion is of physiological interest, as according to the most generally accepted view phosphates are excreted by the kidney in the same way as water, chlorides, bicarbonates and other salts and not as phenolsulphonephthalein.[7]

The phenolsulphonephthalein excretion does not run completely parallel to the phosphate excretion in those animals in whom repeated administration of the latter leads to an increase in phosphate excretion. But in general I have found the phosphate test a more specific one than the phenolsulphonephthalein test in early renal insufficiency as shown in moderately advanced conditions (see Charts III and IV) when the phosphate excretion is much below normal, while the phenolsulphonephthalein is still within or close to the normal range. In advanced stages of renal insufficiency phosphate excretion and phenolsulphonephthalein excretion yield about the same percentage, the phosphate being rarely more than 10% above the phenolsulphonephthalein excretion. In the normal animal, however, phosphate excretion runs about 20% above the phenolsulphonephthalein excretion.

Severe damage might be expected after repeated administration of inorganic material and at present the prevalent view seems to be that phosphate ingestion is harmful when nephritis is already present. Hirsch[8] has shown that the subcutaneous administration of phosphates in rabbits produces necrosis of the cells lining the convoluted tubules and the loop of Henle. The doses given by Hirsch were massive, at least ten times greater than those I used and they consisted of either monosodium or disodium phosphate alone. After injection the rabbits had a severe intoxication followed by death. This effect was not seen in my dogs even after very numerous injections. The dogs remained normal except for occasional albuminuria which may nevertheless be taken as a sign of renal irritation by phosphates. Since my dogs were apparently unaffected by many injections of a larger dosage, I feel it is quite reasonable to administer 350 milligrammes of phosphorus as sodium phosphate for the purpose of a renal function test in experimental work. The anatomical changes immediately following injection were not studied, but normal animals were examined after death a fortnight or later after the last of a long series of phosphate injections. In such dogs changes similar to those described by Hirsch were not found.

Of the six animals with renal lesions, which received phosphate injections, three had a serious nephritis. The first dog in this group (Table III) ran the expected downhill course and the phosphate injections do not appear to have had any decided influence. The rather sudden death is accounted for by the complicating pneumonia.

The next two animals (see Charts I and II) were both advanced nephritics in whom a steady increase in nitrogen retention coincident with the general progress of the disease might have been expected. In neither case was this expectation fulfilled as in both these animals the renal function appeared to be stationary for six months after phosphate injections were started. A remarkable fact was the physical improvement, which occurred during the phosphate injections, as shown by the increase in weight and the cessation of vomiting.

The phosphate solutions given to these two animals were of different hydrogen ion concentration. The dog (see Chart I) which received phosphates pH 7·1 to 7·2 was able to maintain a normal acid-base balance; the dog (see Chart II) receiving phosphates pH 7·4 to 7·6 had a persistent acidosis. It has already been noticed in dogs with advanced X ray nephritis that after the appearance of definite acidosis the alkali reserve rapidly falls to fatal limits and a similar train of events was anticipated as the phosphate-injected animal reached a low level of carbon dioxide combining power (below 25%); but at this unusual acid-base balance the dog lived for five months. At the same time the calcium content of the blood serum was elevated.

I felt that the long standing elevation of the serum calcium in connexion with the elevated blood phosphates may possibly offer some explanation for the hæmorrhagic diathesis as observed in this animal as well as in another untreated one. Collip[9] has produced similar hæmorrhages in normal dogs by administration of single massive doses of combined calcium and phosphate salts or by injection of parathyreoid extract.

The dog which received the larger amount of acid phosphate, developed a large calculus in the renal

pelvis. This stone was found on chemical analysis to be largely composed of phosphate. Both animals (see Charts I and II) had areas of peculiar salt deposit in the basement membranes of the tubular walls and in the capsules. These peculiar salt deposits are quite different from those frequently seen in kidneys of dogs with untreated X ray nephritis.

The remaining three of the six dogs had moderate renal lesions when the phosphate injections were started. No dog of this group died of renal insufficiency even after receiving more than fifty injections over a period of a year. On the contrary they showed a slight improvement, by the results of the functional tests and in their general health.

I am inclined to attach some significance to this observation, as in a series of thirty-two dogs with X ray nephritis of all degrees none showed any improvement for periods greater than two months.

I am not prepared to offer a final explanation for the mechanism of this therapeutic action of phosphate injections, but I think that in all probability a number of different interdependent factors may be responsible.

The value of phosphate administration with respect to general health and body resistance under trying conditions has been pointed out by Embden and his school. Recently Shurley[10] has shown in the case of the normal rat that phosphate feeding may act as a mild stimulant to spontaneous activity. My nephritic dogs which had had no phosphates injected, frequently developed grave signs of malnutrition. This condition in turn undoubtedly had a bad influence on their nephritis, setting up a vicious cycle. Many of the untreated animals also became listless and disinterested in their surroundings, were unclean and their coats became thin and dull. The injected animals offered a striking contrast. They were lively and in remarkably good condition up to the terminal stage of the disease. Their coats were thick and glossy.

According to the work of Haldane, Wigglesworth and Woodrow[11] and other authors the excretion of acids by the kidneys is greatly facilitated by the presence of a large amount of phosphate buffers. This may help to explain the fact that although severe acidosis was present, fatal levels were not reached for many months during treatment (see Chart II). The diuretic effects of phosphate injections were almost invariably noted and much speculation is possible along this line. From later experiments I am inclined to think that certain types of more or less continuous strain on the still functioning renal substance of the fibrotic kidney prolong its efficiency, most probably by hypertrophic processes. Further work is needed to confirm this hypothesis.

Finally it is realized that the series of nephritic animals treated with phosphates is too small to provide definite conclusions regarding the therapeutic action of phosphate injection. Only in one instance was it possible to set up a direct control on an animal of the same breed (see Chart III). Unfortunately the disease was produced by a different technique in different animals; for example in some the kidneys were exposed directly to X rays, in others, the exposure took place through the abdominal wall. For this reason an attempt is now being made to produce a standardized X ray nephritis in which the expectation of life will be better known.

Clinically I think it would be justifiable to give phosphates intravenously for diagnostic or therapeutic purposes to certain selected groups of nephritic patients. Reference is made to the injection of buffer solutions as described from a somewhat different point of view by Hecker.[12]

In a preliminary communication[13] I published many of the experimental details of the work. After this paper had been submitted for publication an article by Brain and Kay[14] became available. Their findings agree with my experimental observations.

Conclusions.

1. Intravenous injection of certain quantities of sodium phosphate into dogs has been found to give valuable diagnostic and prognostic information.
2. The ill-effects noted even from copious doses are not sufficient to discourage the application of this phosphate test in experimental or clinical work.
3. In nephritic animals the phosphate injections seemed to exert a beneficial therapeutic action in regard to the course of nephritis, particularly the nutritional condition.

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HYPEREMESIS GRAVIDARUM: WITH SPECIAL REFERENCE TO TREATMENT BY GLUCOSE AND "INSULIN."[1]

By CONSTANCE E. D'ARCY, M.B., Ch.M. (Sydney),
Lecturer in Clinical Obstetrics, University of Sydney; Honorary Surgeon, Royal Hospital for Women, Sydney; Honorary Gynæcologist, Saint Vincent's Hospital, Sydney.

THE toxæmia of pregnancy manifests itself in many ways and to many degrees, but no form has given the medical attendant so much genuine anxiety and distress as that which produces hyperemesis. In the past cases have continued to occur in which the patient has vomited and continued to vomit in spite of all treatment, unless the uterus was emptied early enough and notwithstanding the emptying of the uterus in hyperemesis at or near term, the patient has become jaundiced, shown acetone, diacetic acid and later albumin and casts in the urine, become cyanosed, comatose and has died. Recent researches into the chemistry of the blood in normal and pathological pregnancy have focused attention on the variation of blood sugar in these conditions and have led to the treatment of the toxæmia, more especially those forms associated with vomiting, with dextrose and "Insulin" and most encouraging reports have been published. I propose to give my personal experiences of using this method of treatment and also a short *précis* of recent literature on the subject.

It is always difficult to judge results of any given method of treatment in a published series of cases, because the severity and degree of toxæmia are not known. We know that many patients with hyperemesis get better as soon as they come into hospital, with ordinary simple, hygienic rules. To quote the Rotunda Hospital report of last year:

> There were no cases of hyperemesis of any consequence. A number of women were admitted at various early stages of pregnancy with excessive vomiting, but all responded to a routine treatment of starvation for twenty-four hours, enemata followed by purgatives, and left hospital well, after regulation of diet and bowel function.

If such cases as these had been treated by glucose and "Insulin" or any other new treatment, they would have made a handsome addition to the list of recoveries. I do not propose to speak of this class of case in any detail. I believe they are all toxic, and regret that text books still continue to divide hyperemesis into two types, the toxæmic and neurotic. This classification gives students and young graduates a wrong impression, and I believe that many patients have been allowed to slip too far by inexperienced medical advisers, because the neurotic element has been over stressed and given too much weight in the judgement of the medical attendant. It is a fact that nervous instability is more manifest in some types of women than in others, but this should be regarded as a symptom rather than a cause.

In mild hyperemesis the general method of treatment I adopt is, in the first place, to put the patient to bed, if possible out of doors on a balcony. A ward where patients are vomiting after an anæsthetic is the worst possible place for a patient suffering from the vomiting of pregnancy. Hospital patients are usually constipated, so an enema is given and a dose of calomel at night and, if tolerated, a saline in the morning. The patient is encouraged to drink orange drinks with glucose, barley water with fruit juice and water. Should she continue to vomit, a 5% solution of glucose in saline solution is administered *per rectum*, usually three hundred cubic centimetres (ten ounces) every four hours or, if better tolerated, by the continuous Murphy's drip method. If she salivates, atropine 0·54 milligramme (one one hundred and twentieth of a grain) is given every eight hours. Nearly all medicine by mouth nauseates, more especially when the substance is gritty by nature. An alkaline mixture containing a soluble form of bismuth is sometimes beneficial and well tolerated. Toxæmia causes acidosis and the vomiting and consequent starvation increase the acidosis. Rest in bed with freedom from anxiety for a few days, together with absorption of fluid and glucose will put right the patient with average mild hyperemesis. A patient with a more obstinate form is sometimes enabled to carry on her pregnancy by the administration of extract of *corpus luteum*. I use the extract put up in ampoules by Parke, Davis and Company, and give one ampoule by intramuscular injection night and morning. I have not given *corpus luteum* intravenously, though the method is commonly mentioned in American literature.

A recent case is interesting.

> A young bride developed symptoms of severe acidosis in the first weeks of her pregnancy. I carried out treatment in hospital with glucose, saline solution and fruit juices and *corpus luteum* as indicated. She made rapid progress. The injections of *corpus luteum* were stopped when she got up to the stage of eating chicken, fish, bread and butter, puddings *et cetera*, and all trace of acetone had disappeared from the urine. She commenced to vomit again, but recovered under the same treatment. This series of incidents happened twice and so the *corpus luteum* administration was continued until the third month, when she developed immunity to the toxin.

If symptoms do not subside, the deficiencies in food, fluid and salts must be supplied, or the patient will die. Intravenous injection of glucose in salt solution has been used in these cases for some years. In my own experience intravenous injections of glucose without "Insulin" will not benefit a patient severely ill. I will briefly quote three cases:

> Patient A was seen by me in consultation four years ago. Her medical attendant informed me that the patient had a normal pregnancy and came into labour at term. She was a *primipara* over thirty years of age. There was

[1] Read at a meeting of the Eastern Suburbs Medical Association, January, 1929.

some vomiting during labour, but not sufficient to cause comment. She had a long labour and chloroform inhalations were given to allay her suffering. Delivery was instrumental, long and difficult and was effected under chloroform anæsthesia. The urine manifested no abnormality. The second day after delivery she showed signs of drowsiness and vomiting. When I saw her on the third day she was comatose, deeply jaundiced, with a bluish tint in her prominent features and in her extremities. The urine contained a heavy deposit of albumin, acetone, blood and bile. Injections intravenously and by rectum of a 10% solution of glucose made no difference to her condition and she died within twenty-four hours.

Patient B had a somewhat similar history. She was a young *primipara* with a slightly generally contracted pelvis who went through a normal pregnancy, except that she had a fair amount of vomiting in the early months. She came into labour in response to quinine stimulation shortly after the estimated date of term. Her medical attendant informed me that she had a long labour and a very difficult forceps extraction with considerable laceration of the perineum. Chloroform anæsthesia was again used. The urine manifested no abnormality. The second day after delivery she began to vomit, the colour of the vomit being dark, so-called coffee grounds in nature, her pulse was rapid and she was restless and mentally unstable. On the third day, when I first saw her, she was deeply jaundiced, the skin was cold, pulse soft and rapid and she was vomiting more or less constantly. She had a large hæmatemesis on the night after the third day and continued to vomit bright blood on and off till she died. The urine was bile stained and contained much albumin, acetone and some blood. She had intravenous and rectal injections of glucose, but died on the fourth day.

Undoubtedly, both these patients had delayed chloroform poisoning, as well as pregnancy toxæmia. These two cases give a lesson on the long continued use of chloroform during labour.

Patient C had had three children. She had been vomiting more or less persistently since pregnancy began. She was five months pregnant when admitted to hospital and in addition to excessive vomiting, she had pyelitis. Both conditions began to yield to treatment. She was ravenous for food and took large quantities of varied foods brought in by friends, when she was not under strict observation. She began to vomit again. She was seemingly retaining a fair amount of food, but she was suffering from one of those deceptive conditions in which acidosis developes insidiously and in which the patient slips before one is aware of the danger. She was apparently in fair health and spirits when she began to have hallucinations and show signs of mental restlessness. This is always a grave danger signal. As these symptoms occurred at night, they were unfortunately not reported. In the morning, she was cold, blue, comatose, with running pulse. The urine contained acetone, although previously clear. Intravenous and rectal injections of glucose did not prevent a fatal end on the same day.

In the light of present experience I believe all these three patients would have lived if "Insulin" had been given as well as intravenous injections of glucose.

In a paper published in the *American Journal of Obstetrics and Gynecology*, January, 1927, E. S. Waters[1] gives the results of treatment of eighteen patients with hyperemesis:

Thirteen were treated with high carbohydrate diet, saline hyperdermoclysis, sufficient fluid intake, *corpus luteum* intravenously and symptomatic treatment of various kinds before intravenous injection of glucose and insulin were used, with resulting cure. There was one recurrence which responded to treatment. The entire series he would classify as "severe or pernicious" for exhaustion, emaciation, dehydration, beginning nephritis or early jaundice were singly or severally present and acidosis was present in every case. One injection caused cessation of vomiting in 14 patients within twenty-four hours and the average duration was 13 days.

W. E. Levy[2] used glucose and "Insulin" for six patients with vomiting of pregnancy, all with low blood sugar and low carbon dioxide combining power of the blood, as well as acetone in the urine in four instances. In no instance was abortion necessary; four of the patients were subsequently delivered at term and the other two were progressing normally when they were last seen.

W. J. Dieckmann and R. J. Crossen,[3] writing in the *American Journal of Obstetrics and Gynecology* of July, 1927, affirm that vomiting of pregnancy is due to a deranged metabolism of the maternal organism, particularly of carbohydrates. They state that alkalies in treatment are not only not indicated, but are actually dangerous. They point out that experimental work indicates that "Insulin" with glucose given intravenously replaces glycogen stores much more quickly than intravenous injections of glucose alone, and, if true, "Insulin" with large amounts of glucose may give better results than glucose alone.

The results in a series of 48 cases since 1921, with none requiring abortion, show that the treatment of this condition has been greatly changed since the continuous use of large amounts of intravenous glucose solutions have proved so successful.

P. Titus and P. Dodds[4] give intravenous injections of glucose solution as a routine after major obstetric and gynæcological operations as a preventive of shock and distension, nausea and acidosis. Sometimes there is a reaction, manifested by a sharp chill, usually with considerable rise of temperature, with shock and collapse. They hold that reactions are due to faulty technique, in preparation of the glucose solution or the method of administration.

The following rules for dosage are quoted from De Lee's extract in the "Practical Medicine Series" (Obstetrics and Gynæcology) of the paper by Titus and Dodds:

1. *Dosage*: The average dose for an adult is about one gramme per kilogram of body weight. An average adult weighs between 50 and 70 kilograms, so that it was decided to establish 75 grammes as the initial dose, subsequent doses to be 50 grammes.

2. *Glucose* (Dextrose): The glucose should be chemically pure. Several different preparations have been used, and while there is little choice, if chemically pure, the authors usually use Merck's c.p. anhydrous glucose.

3. *Solvent*: The glucose should be dissolved in freshly double distilled uncontaminated water, and not in salt solution, or sodium bicarbonate solution. In the preparation of a single dose, 50 grammes of the glucose are carefully weighed and then dissolved in 200 cubic centimetres of freshly distilled water, using only glassware which has been thoroughly washed in distilled water. Before preparation for sterilization the solution should be filtered at least five or six times to remove even tiny particles of dust or cotton fibres.

4. *Concentration of Solution*: The authors emphasize that a 25% solution (50 grammes in 200 cubic centimetres of water) is the generally desirable strength. It is a distinct advantage to use a strongly hypertonic solution, because its hypertonicity actually favours a more rapid interchange between the tissues and the blood stream, so that toxins are diluted, œdema lessened, and the sugar

seized and stored more rapidly by the tissues. If more fluids are required it is safe to consider this as an entirely separate matter, and administer them as sub-mammary infusions of salt or weak glucose solution.

5. *Sterilisation*: The flasks of glucose solution, after being properly stoppered with cotton plugs in gauze, and the top sealed with lead foil, should be sterilised in a steam steriliser for half-hour at 100° C. on three successive days. For emergencies the flask may be sterilized in an autoclave at 15 pounds pressure for 20 minutes. Solutions showing carmelisation or sediment after sterilisation should be discarded.

6. *Age of Solution*: Glucose kept in flasks stoppered with cotton, or cotton and gauze, and sealed with lead foil, will show comparatively little change in hydrogen-ion content over a considerable period. Solutions should preferably not be kept longer than four weeks unless hermetically sealed.

7. *Ampoules of Glucose Solution*: Usually 50% solutions are now on the market with directions for dilution. (Such ampoules put up by Parke, Davis and Co. may be bought in Sydney now.)

8. *Technique of Administration*: The injection should proceed slowly, at the rate of 4 cubic centimetres per minute for a 25% solution, and during this period the solution should be kept hot. The solution is ordinarily injected from a salvarsan tube, using a small calibre needle, and having the tube coiled in a basin of hot water to keep the temperature of the flowing fluid up to 100° to 110° F. Fresh rubber tubing should not be used until after it has been thoroughly washed in running water, then boiled in clean water (without sodium bicarbonate), again washed, and finally sterilised in an autoclave. When merely boiled sediment from the water is often to be seen within the glass. Before beginning the injection the system should be rinsed out by running distilled water through the salvarsan tube, the rubber tubing, the connections, and the needle.

I will conclude by giving the details of a recent successful case in my own practice.

E.B., aged twenty-seven years, was discharged from hospital two weeks previously. She had been treated for *hyperemesis gravidarum*. When discharged she was walking, was on full diet and feeling very well. She commenced vomiting two days after leaving hospital and the vomiting steadily became worse, until at time of admission to the Royal Hospital for Women on July 17, 1928, she was not even keeping sips of water down. She had lost 12·6 kilograms (two stone) in weight in two weeks. She complained of headache, the bowels had not been opened for some days, she had no œdema and no urinary symptoms. On examination she was seen to be very ill. The temperature was 36·1° C. (97° F.), the pulse 120 and very soft. The sclerotics were deeply jaundiced. She was vomiting a coffee grounds substance almost constantly. She manifested mental restlessness and a tendency to delirium. She was very tender over the lower part of the abdomen, but no rigidity was noted. *Per vaginam*, the uterus was found anteverted and enlarged to the size of three months' gestation. A catheter was passed and four ounces of urine withdrawn from the bladder. The urine was acid, its specific gravity 1,010, it contained about one tenth albumin on being boiled, acetone was present, but no sugar, no pus nor blood. A centrifugalized specimen of urine contained epithelial casts. The blood urea was sixty-three milligrammes per hundred cubic centimetres of blood and the specimen of blood taken was later reported to give a partially positive Wassermann reaction. Unfortunately, no estimation of blood sugar was made. She was given a bowel wash-out and rectal injection of 5% solution of glucose which she did not retain. At 2 p.m. her condition became very bad, her pulse was very rapid and soft, indeed barely palpable. Her colour was blue, her skin was cold and she was semi-conscious, with continuous hiccup, with occasional ejection of dark blood-stained vomit. Strychnine and digitalin were injected and preparation made for intravenous injection of glucose. At 6 p.m. she was given 568 cubic centimetres (one pint) of 10% solution of glucose in normal saline solution by intravenous injection and at the same time thirty units of "Insulin" by hypodermic injection. By the time this was commenced she was quite comatose. Almost immediately she showed signs of improvement and in half an hour was warm to touch, conscious, talking and the pulse easily palpable. The temperature went up to 37·2° C. (99° F.) and she was mentally clear. I had noticed this immediate dramatic improvement before in a similar patient who began to move restlessly and to talk and to swallow immediately after and even during the injection of the same quantity of a 10% solution of glucose in saline solution, accompanied by twenty units of "Insulin." But in the latter case the improvement was not maintained. Could the smaller dose of "Insulin" account for this? About a litre of saline solution was given under the breasts at 8 p.m. Her condition had definitely improved, the temperature being still 37·2° C. (99° F.), the pulse 124 in rate and of fair quality and the vomiting less.

At 11 p.m. she was very restless, trying to get out of bed; the extremities were cold, the pulse rate 160. A hypodermic injection of morphine 0·015 gramme (one-quarter of a grain) was given.

On July 18, 1928, at 10 a.m. her condition was very bad. She was very cold, her temperature 36° C. (96·8° F.) and it was impossible to feel any pulse. The tongue was brown and dry with sordes about the mouth. The breathing was very shallow and at times seemed to cease altogether. A second injection of 568 cubic centimetres (one pint) of 10% glucose solution in saline solution and a hyperdermic injection of thirty units of "Insulin" were given. My house-surgeon, Dr. Ross, gave this injection. He had to cut down on the vein to get the needle in, as the veins were too flat and empty. She did not feel the cutting or manipulations. The hand over the heart could feel no pulsation and for a short period we could not see any movements of respiration. We thought she had died. We carried out the movements of artificial respiration and she revived. Again, there was the same dramatic improvement as soon as the injection was finished. The time taken for the injection was about thirty minutes. She was given further submammary injections of saline solution. A drip saline and glucose (5%) injection was started *per rectum* by the Murphy method. She was sensible all day and helped materially in the treatment by keeping still. At 5 p.m. her condition was much improved. She had retained a litre and a half of glucose and saline solution given *per rectum*. However, she secreted only sixty cubic centimetres (two ounces) of urine in the twenty-four hours. The urine was acid, its specific gravity was 1,010; albumin was one-tenth when the tube was allowed to stand after bottling the urine; acetone was present to a slight extent, there was no sugar, no pus and no blood. At 8 p.m. she was restless; there was difficulty in keeping her in bed and she was no longer retaining the rectal injections. She vomited small mouthfuls of dark blood-stained fluid only occasionally and retained some jelly, orange juice and water.

On July 19 she was in a state of constant hiccup, but not vomiting and was taking drinks in small quantities and jelly. She secreted no urine at all this day. A second estimation of blood urea was made and the figure was ninety-six milligrammes per hundred cubic centimetres of blood. Her pulse was soft and toneless, varying from 120 to 136, the temperature subnormal. The low specific gravity urine with epithelial casts and the high blood urea seemed to indicate chronic nephritis and she had apparently come to the verge of uræmia. It seemed as if there would be no hope at all of her living without induction of labour, whereas there might be some chance of her kidneys regaining their function if the uterus could be emptied. Dr. Hipsley saw her in consultation and we agreed to give her this chance. Accordingly sea-tangle tents were inserted into the *os* without anæsthetic and the vagina packed with gauze.

On July 20, 1928, her general condition showed slight improvement, but she had intractable vomiting and hiccup. She secreted no urine again for a second period of twenty-four hours. She was given five doses of pituitrin in 0·18 mil (three minim) doses, at intervals of half an hour to assist the uterine contractions. The tents were not

expelled, so at 2 p.m. Dr. Williams, the Medical Superintendent, removed the tents and gauze and, finding good dilatation, was able to remove the uterine contents and give an intrauterine douche. No anæsthetic was used. A light gauze packing was left in the uterus and one cubic centimetre of pituitrin was given immediately after the operation which she stood well.

On July 21, 1928, she passed practically no urine all day; the skin was dry and her condition was marked on the chart as only moderately good. In four days she had secreted 180 cubic centimetres (six ounces) of urine. It was decided to give her a hot pack. She stood this well and whether it was cause and effect or coincidence, she began to secrete urine and 330 cubic centimetres (eleven ounces) were drawn off by catheter. The urine had a specific gravity of 1,020, the albumin was one-tenth, acetone was present, but no sugar, no pus, no blood were found.

At 10 p.m. she vomited a large clot of blood. The hiccup became less frequent, her general condition was improving. She was given morphine 0·015 gramme (one-quarter of a grain) as she was very tired and not inclined to sleep.

On July 22, 1928, she was passing a moderate amount of urine into the bed with stool. Three hundred and thirty cubic centimetres (eleven ounces) were withdrawn by catheter. The urine was acid and had a specific gravity of 1,020, albumin was one-tenth, acetone was present, but no sugar, a trace of pus was found, but no blood. The temperature was 36·1° to 36·7° C. (97° to 98° F.); the pulse rate was 120 to 130. She complained of severe abdominal pain and of a feeling of pressure over the bladder. She was tender over the lower part of the abdomen, but not on the uterus. The signs and symptoms suggested intraabdominal hæmorrhage. (Examinations made *post mortem* in this condition frequently reveal even large hæmorrhages into the abdomen; one of two litres was mentioned in the Rotunda Hospital Report of 1925-1926. Hæmorrhages are also noted in the endothelial lining of the heart, into the liver, kidneys *et cetera*.)

On July 25, 1928, she had passed two litres of urine in the previous twenty-four hours. The general condition showed definite improvement. The urine had a specific gravity of 1,014, was acid, but contained no albumin, though acetone was present; the temperature was 36·1° to 36·7° C. (97° to 98° F.). The pulse rate was 120 to 130.

On July 27, 1928, the urine was free from acetone; she passed a litre in twenty-four hours; its specific gravity was 1,010 and a trace of albumin was present. She ceased vomiting and was taking copious fluids and a reasonable amount of solid food. She complained of dimness of vision. The ocular fundi were examined by Dr. Granville Waddy, who reported "both ocular fundi normal."

On July 28, 1928, the urine measured 2·1 litres (seventy ounces), a trace of albumin was present, but no acetone. The blood yielded a partial positive response to the Wassermann test.

On August 8, 1928, another estimate of blood urea resulted in a finding of forty-six milligrammes per hundred cubic centimetres of blood. She was anxious to go home and went home on August 12, 1928, walking out of hospital.

This patient was not only the subject of hyperemesis, but also of severe kidney disease. If the latter condition had not been present, I have no doubt that she would have recovered without interruption of the pregnancy after the glucose and "Insulin" injection. Her appearance, indeed, was almost like a resurrection. The vomiting and hiccup after this stage were uræmic and although acetone was present in the urine it was only just demonstrable.

In future cases I shall use a stronger concentration of glucose solution. I believe that each patient must be considered individually in regard to repetition of doses. The fact that the glucose solution was absorbed so well from the Murphy's drip in my patient, saved further repetition of intravenous injections. In patients of this kind intravenous medication is not easy. The veins are so collapsed and elusive and as the patient comes out of coma she is restless and anæsthetics are undesirable on account of increased risk of acidosis.

In regard to the dosage of "Insulin," H. J. Sander[6] in a paper embodying clinical and experimental studies on the toxæmias of pregnancy, states:

> Insulin, with a protective dose of glucose approximately two grammes per unit of insulin, has proved to be satisfactory therapy in cases of acidosis.

On inquiry in Sydney hospitals I find that the average doses adopted in cases of acidosis from causes other than the toxæmias of pregnancy, for example, post-anæsthetic acidosis, are in one hospital glucose up to fifty grammes with "Insulin" at the rate of one unit to each 1·5 grammes of glucose. At another hospital where the imperial system is adopted, glucose in 10% solution is made and "Insulin" at the rate of one unit of "Insulin" to each thirty cubic centimetres (one ounce) of 10% glucose solution, twenty units to 568 cubic centimetres (one pint) of glucose solution.

The glucose used in this case was supplied by Elliott Brothers and was imported from Buffalo City.

These patients are eminently suitable for hospital treatment, for they need to be under the continual supervision of a medical officer.

Acknowledgements.

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A FORM OF TYPHOID FEVER FOUND IN NORTH QUEENSLAND.

By A. H. BALDWIN, M.B., B.S., D.P.H. (Melbourne),
Acting Director, Australian Institute of Tropical Medicine,

AND

A. J. BEARUP,
Senior Technical Assistant, Australian Institute of Tropical Medicine, Townsville.

IN Northern Queensland far more than in the non-tropical part of Australia the practitioner is

apt to meet with fever in a patient and to be uncertain as to its cause.

In former times the diagnosis of malaria was often applied to many fevers of uncertain ætiology and in more recent times the terms "low fever" and "coastal fever" have also been so applied.

From this conglomerate group of fevers Lilley and Bearup,[1] of this Institute, were able to split off a specific fever group and show that it was due to an infection with *Bacillus pyocyaneus*. Still later another group was shown by agglutination tests to be endemic typhus. The present note has been written to show how a still further group may be separated from these undiagnosed fevers. The result of the work performed also again emphasizes the paramount importance of early blood culture in all tropical fevers.

From time to time reports by different writers indicated that what were considered typical clinical cases of typhoid often yielded no reaction to the Widal test.

Priestley[2] called attention to this fact repeatedly in half-yearly reports from this Institute thus:

> Widal reactions irregular and only typical in a few cases.
>
> The anomalous results obtained with the Widal reaction in cases of typhoid . . . .

W. E. George, July, 1926 (in official correspondence) reported a succession of failures to react to the Widal test. A patient from whom one of these sera had been obtained, was found at autopsy to have a typical perforation and ulcers in the intestine.

K. C. Ross[3] reported an interesting case of typhoid in which repeated agglutination tests yielded no reaction.

A. B. Lilley[4] read before the New South Wales Branch of the British Medical Association an account of an atypical form of typhoid bacillus which he had met with at the Townsville General Hospital.[1]

The intention of the present article is to draw attention to the fact that these peculiar cases of typhoid are not isolated examples, but that there exists in Northern Queensland at any rate, a form of typhoid which fails to give a positive reaction to the Widal test when standard typhoid antiserum (Rawlings) is used. Furthermore it would seem from the evidence at our disposal that in any outbreak due to this particular strain of typhoid organism this failure to agglutinate with antiserum (Rawlings) is a constant feature in most cases belonging to that particular outbreak.

Our attention was first attracted by the circumstances attending the illness of a patient in the Townsville General Hospital. In February, 1928, a patient in that institution suffering from grave malignant disease, developed pyrexia. His serum was tested and failed to yield a reaction to the Widal test. An organism, however, was cultivated from his blood with the morphology, staining reactions, sugar reactions and agglutination reactions of *Bacillus typhosus*. Repeated examinations of this patient's serum gave no reactions to the Widal test. The reliance to be placed on absence of reaction under the circumstances was problematical. (Lilley's case cited above.)

A short time later medical practitioners in Cairns after having received reports that certain sera which they had sent to this Institute for examination yielded no reaction with the Widal test, made the diagnosis of "coastal fever" in certain instances. Then one patient died, a very rare event in true "coastal" or "Mossman" fever. An autopsy was performed and a perforation with typical typhoid ulcers in the intestine was discovered.

The position was no doubt rendered more difficult by the fact that typhoid fever, with the exception of a couple of imported cases, had never occurred in Cairns. Furthermore, the clinical picture of typhoid fever, as we see it in North Queensland, is very different to that described in text books or as met with in southern Australia. It is a milder disease; diarrhœa is often not pronounced, indeed the attack often starts with constipation. The rash is generally very poorly developed. The spleen is very variable and its enlargement is not so pronounced as text books describe. The mortality is low.

The death of the patient at Cairns and the occurrence of further cases led to the appearance in the local and other papers of articles in which the outbreak was generally referred to as "The Mysterious Disease at Cairns." When this Institute had reported a number of specimens of blood as yielding no reaction to the Widal test, fæcal specimens were asked for that this result might be checked. In addition a request was sent for further specimens of serum.

From the fæcal specimens forwarded a bacillus in every way resembling *Bacillus typhosus* was cultivated, but the sera still failed to react to the Widal test.

At this stage, at the invitation of several Cairns practitioners, it was decided to investigate the phenomenon on the spot. Unfortunately only two days could be spared for the trip and the fact that at this time Cairns possessed no laboratory, imposed considerable technical difficulties in the way of working out the problem.

Laboratory Investigations.

On account of the tediousness and complexity of the practical work full details will not be given, but briefly the work done on the outbreak at Cairns was as follows.

Fæcal Cultures.

Eighteen fæcal specimens were collected and cultured. Typical colonies were subcultured next day and after six hours' incubation transported to this Institute.

Three of these gave growths of an organism with the morphology, sugar and staining reactions of

[1] A full account of Dr. Lilley's report appears in another place in this issue.

ILLUSTRATIONS TO THE ARTICLE BY DR. ADOLPH BOLLIGER.

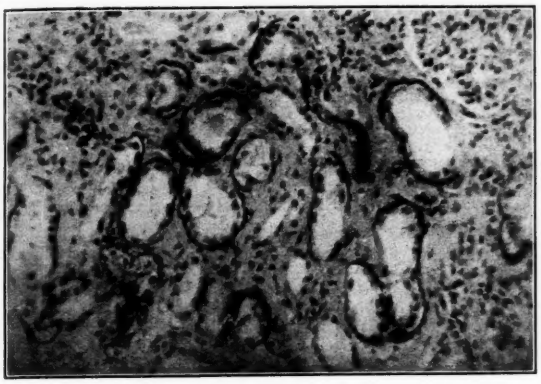

FIGURE I.
Salt Deposits in the Basement Membranes of the Tubular Walls.

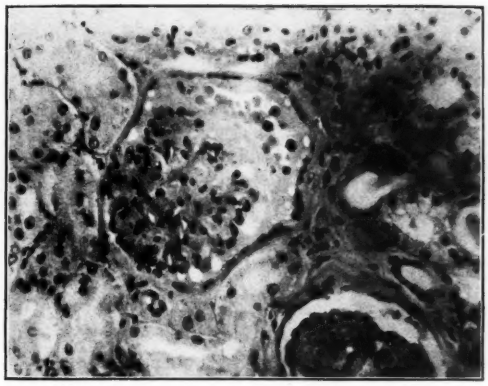

FIGURE II.
Small Salt Deposits in a Fibrosed Area and in the Capsular Space of an Atrophic Glomerulus.

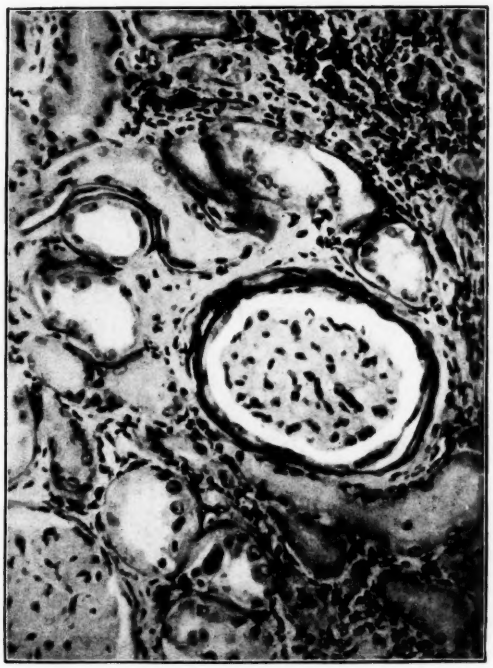

FIGURE III.
Salt Deposits in the Capsule of Bowman and in the Basement Membranes of the Tubules.

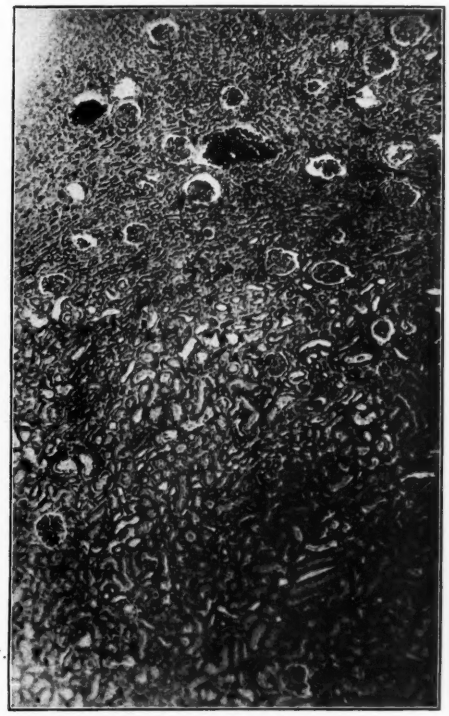

FIGURE IV.
Transitional Area between almost Completely Fibrosed Tissue and a Pole with Approximately Normal Structure. Note the salt deposits in the fibrosed area.

ILLUSTRATIONS TO THE ARTICLE BY DR. ADOLPH BOLLIGER.

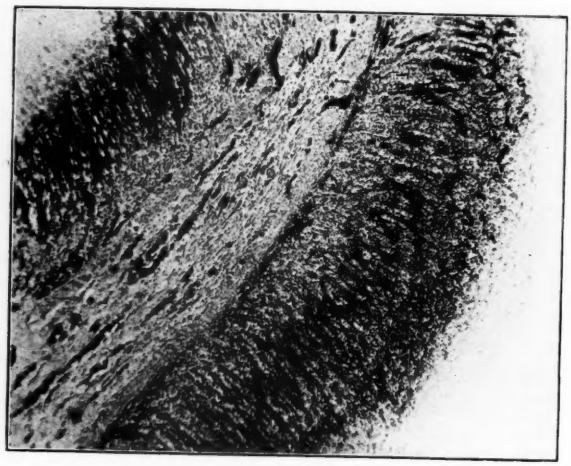

FIGURE V.
Numerous Hæmorrhages in the Stomach Wall.

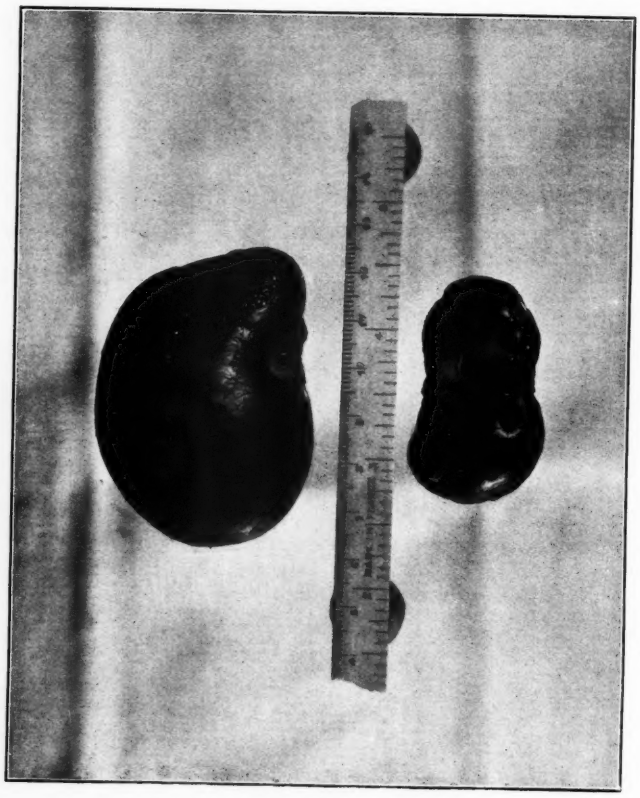

FIGURE VI.
Right Kidney Showing Very Marked Degree of Contraction and Fibrosis.

Bacillus typhosus. This organism agglutinated in high titre with the serum of the patient from whom the specimen had been obtained, with other "Cairns" sera and with stock antityphoid serum (Rawlings).

Blood Cultures.

Four specimens of blood were cultivated and all were positive. The organisms cultivated were similar in their morphology and behaviour to those cultivated from the fæces.

Urine Cultures.

Six specimens of urine were cultivated and a similar organism obtained from one.

Agglutinations.

Blood for the Widal test was taken from seventeen patients. The Widal test was performed with two emulsions, one of the "Cairns" organism and one of typhoid (Rawlings).

The result of the Widal test was as follows: Agglutination, sixteen; no agglutination, one (to both emulsions). Among the sixteen positive results "Cairns" emulsion gave better agglutination than "Rawlings" in eight. "Rawlings" emulsion gave better agglutination than "Cairns" in none. Equal agglutination occurred with both in five. "Cairns" emulsion caused agglutination, but "Rawlings" emulsion did not in three. "Rawlings" emulsion caused agglutination, but "Cairns" emulsion did not in none.

Considering those five sera which gave an equal agglutination with both emulsions, if we take note of heavier agglutinations in lower dilution than one in forty then only one serum gave as good an agglutination with "Rawlings" as with "Cairns" emulsion.

The "Cairns" organism was then tested against stock sera of the following: Shiga, Flexner, Aertrycke *A* and *B*, cholera, paratyphoid *A* and *B*. Read after two hours all tubes manifested no agglutination.

Read overnight Aertrycke *B* gave agglutination in 1:40 and a slight trace in 1:80.

"Cairns" organism and "Rawlings" emulsion were then agglutinated against stock antityphoid serum (Rawlings). Both gave a triple positive agglutination with 1:5,000.

Absorption Tests.

We were almost certain now that we were dealing with a typhoid organism behaving in a peculiar way in agglutination tests. To prove that this was so, it was necessary to carry out a series of absorption agglutination tests. The method used was that recommended by the Medical Research Council.[5]

We were particularly fortunate in so much as we encountered one patient, number 19, who yielded a blood serum with the extremely high natural titre of +++ 1/1280 ++ 1250 against the "Cairns" organism. We were thus enabled to use this serum in our absorption tests.

(*a*) "Cairns" serum diluted 1:10 absorbed with "Cairns" organism; agglutinated against "Cairns" and typhoid.

(*b*) "Cairns" serum diluted 1:10 absorbed with typhoid; agglutinated against "Cairns" and typhoid.

(*c*) Stock antityphoid serum diluted 1:10 absorbed with "Cairns" organism; agglutinated against "Cairns" and typhoid.

(*d*) Stock antityphoid serum diluted 1:10 absorbed with typhoid; agglutinated against "Cairns" and typhoid.

Four similar tests were then done, one-eleventh of the absorbing dose used in the above test being employed. With full absorbing doses the results with the two organisms were identical.

When one-eleventh of the absorbing dose was used (a much more delicate test) the results were so nearly identical that it was clear that "Cairns" was a typhoid organism and not a member of any closely allied group. To confirm this finding "Cairns" serum was absorbed with typhoid (Rawlings) emulsion and agglutinated with the sera mentioned in the accompanying table; the results are shown opposite to each.

Organism.	Agglutination.	
	Two Hours.	Overnight.
Aertrycke *A*	Nil	Nil
Aertrycke *B*	Nil	Trace 1:50
Paratyphoid *A*	Nil	Slight trace 1:50
Paratyphoid *B*	Nil	Nil
Gärtner	Nil	Trace 1:50
Suipestifer	Nil	Nil

To test finally that the "Cairns" organism (number 3) we were using was a suitable one, the following tests were performed. "Cairns" serum (number 19) was absorbed with "Cairns" organism (number 19) and agglutinated against "Cairns" organism (number 19), typhoid (Rawlings) and "Cairns" organism (number 3).

The results of these agglutinations were so close as to indicate the close identity of the organisms and the suitability of number 3 for test purposes.

Cloncurry Outbreak.

Just nine months after the Cairns outbreak we had the truth of our previous work brought home to us in a most graphic way. A very brisk outbreak of typhoid fever commenced in Cloncurry. Very rapidly over seventy persons were affected with a mortality rate of over 10%.

On November 7 seven sera from Cloncurry were examined by us and all gave no reaction to tests for typhoid fever (Rawlings).

Sufficient serum was available in three cases to test against "Cairns" organisms. All three were found positive to this organism (+++ 1:160).

On December 11 a patient arrived in Townsville incubating the fever. A Widal test was subsequently performed. This gave no reaction for typhoid (Rawlings), but a positive one to "Cairns" organism (+++ 1:40). Subsequently an organism

typically *Bacillus typhosus* was cultured from this patient's stool.

Finally, from the urine of a patient who was convalescent in Cloncurry, an organism was cultivated which was typically *Bacillus typhosus*.

Summary.

1. A form of typhoid fever occurs in North Queensland in which the patient's serum either fails to yield a reaction to the Widal test when typhoid (Rawlings) is used or yields only a weakened reaction to this strain late in the disease.

2. Outbreaks of this type have occurred at Cairns and Cloncurry and isolated cases at Townsville, Babinda and perhaps the Atherton tableland.

3. By absorption tests it has been proved that the organism we have termed "Cairns" is a strain of typhoid and not a paratyphoid or member of an allied group of organisms.

4. The occurrence of the strain of typhoid investigated again stresses the paramount importance of early blood cultures in tropical fevers.

5. The work carried out indicates that for the performance of the Widal test in North Queensland an emulsion of "Cairns" should prove of greater value than one of typhoid (Rawlings).

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(1) A. B. Lilley and A. J. Bearup: "Generalized Infections Due to Pseudomonas Aeruginosa (Bacillus Pyocyaneus) with a Study of the Characteristics of Local Strains of the Organism," THE MEDICAL JOURNAL OF AUSTRALIA, March 24, 1928, page 362.

(2) H. Priestley: Australian Institute of Tropical Medicine, Half Yearly Report Ending June, 1914, Half Yearly Report Ending June, 1915.

(3) K. C. Ross: "An Unusual Case of Typhoid Fever," THE MEDICAL JOURNAL OF AUSTRALIA, May 22, 1926, page 580.

(4) A. B. Lilley: THE MEDICAL JOURNAL OF AUSTRALIA, September 1, 1928, page 286.

(5) Medical Research Council, Special Report, Series Number 51, 1920: "The Laboratory Diagnosis of Acute Intestinal Infections, including the Principles and Practice of the Agglutination Test," page 137.

THE PREVENTION AND MANAGEMENT OF PLACENTAL SITE HÆMORRHAGE IN THE HOME.[1]

By A. J. CUNNINGHAM, M.D., Ch.M.,
Sydney.

MY object in presenting this paper tonight is not to submit anything new or revolutionary, but rather to review the recognized methods for the control of *post partum* hæmorrhage (placental site hæmorrhage) and to stress those methods which to my mind are of most value in the home.

The importance of the subject cannot be emphasized too strongly. Apart from the immediate danger to the mother's life from severe blood loss, the risk of puerperal infection is increased to a considerable degree. This is due partly to the lowered resistance of the patient to infection from blood loss and partly to the methods employed for controlling the hæmorrhage.

In the home the risk of infection is heightened by the fact that no matter how much care is taken, surgical asepsis is well nigh impossible.

I propose to describe a plan of action which is efficient and which diminishes this risk of infection to a certain extent.

The Normal Third Stage.

The third stage of labour begins immediately after the child is born and ends with the expulsion of the placenta and membranes. Its duration is from ten to forty minutes, depending on the contractions of the uterus.

After delivery of the child, the uterine contractions begin again and the placenta gradually separates. There is generally some hæmorrhage (about 180 to 240 cubic centimetres or six to eight ounces). When the placenta is separated, it lies in the vagina and lower uterine segment and it is generally necessary to express it. When the placenta is delivered, the uterus contracts firmly and it is felt as a hard mass just above the *symphysis pubis*. A few hours later the uterus becomes softer and larger and the fundus rises to the level of the umbilicus. The separation of the placenta is due to continual retraction of the uterine muscle which acts in two ways: (i) by causing shrinking of the placental site and (ii) by bringing an expulsive force to bear on the placenta.

Two methods of expulsion are described:

1. Schultze's mechanism.—A retro-placental blood clot is formed which separates the placenta above its centre; this clot enlarges and acts as a wedge and the placenta is expelled, fœtal surface first, followed by the membranes.

2. Duncan's mechanism.—The placenta is separated from below upwards and expelled edgeways followed by the membranes.

The Ideal Conduct of the Third Stage.

Tie the umbilical cord. Guard the *fundus uteri* with the ulnar border of the left hand. This must be done by the *accoucheur* himself. No movement is necessary. Watch for hæmorrhage through vaginum from time to time, also watch for accumulation of blood clot in the vagina or uterus. Wait for half an hour by the clock, by which time in 90% of the patients the placenta and membranes will be separated and lying in the lower uterine segment and vagina. The main sign of separation of the placenta is the rising of the *fundus uteri* above the level of the umbilicus and usually to the right of it. The uterus at this time is firmer and smaller than before and does not "feel boggy." There are no indications when the membranes are separated. Knead the uterus into a hard ball and push out the placenta and membranes, the uterus acting as a piston. When the uterus is empty,

[1] Read at a meeting of the Section of Obstetrics and Gynæcology of the New South Wales Branch of the British Medical Association on March 21, 1928.

vigorous massage to the fundus may be indulged in and "Infundin" or liquid extract of ergot given.

Predisposing Causes of Hæmorrhage.

Predisposing causes of hæmorrhage are all those which lead to uterine inertia or atony or interfere with the proper clotting of the blood in the vessels at the placental site. These may be enumerated:

1. General debility of the patient due to some disease peculiar to pregnancy, for example, toxæmia.
2. Over-distension of the uterus.
3. Too rapid succession of pregnancies.
4. Termination of the labour by delivery with forceps in the absence of a uterine pain or when the pains are weakening, that is secondary uterine inertia.
5. Something interfering directly with contraction and retraction. This is generally retained placenta which may be adherent or not. Interference may also be caused by peritoneal adhesions.
6. An unhealthy condition of the uterine wall such as myomata or scars from previous operations.
7. The effects of excessive use of chloroform or hyoscine.
8. Imperfect coagulation of the blood is perhaps an explanation when there is a history of repeated *post partum* hæmorrhage.
9. Abnormal shape of the uterus such as bicornate uterus, *uterus septus* and similar conditions.
10. Displacement of the uterus by a full bladder or rectum. A full bladder is a cause that must be emphasized. It is very easily prevented by making catheterization of the bladder a routine procedure before delivery.

Of these causes the most serious is undoubtedly that due to the termination of labour in the absence of a uterine pain. It is probably the most certain way of producing a *post partum* hæmorrhage.

Post partum hæmorrhage is more common in the hands of medical practitioners than in nurses' hands. This is due to the universal use of chloroform and to the very frequent use of forceps. Delivery of a child without forceps seems to be becoming a lost art amongst medical practitioners.

Actual Causes.

In the vast majority *post partum* hæmorrhage is caused by mismanagement of the third stage of labour. Practically every obstetrician of note throughout the world agrees that severe *post partum* hæmorrhage is undoubtedly the fault of the *accoucheur*. This mismanagement may be due to violent stimulation of the uterus after the baby is born. Perhaps the fact that the uterus relaxes as well as contracts and retracts is forgotten. Especially is this "rubbing up" of the uterus of danger immediately after the birth of the child. An irregular contraction is bound to occur. It may be due to the use of "Pituitrin" or "Infundin" before the completion of the third stage. It may further be caused by premature attempts to deliver the placenta, resulting in a piece of placenta or membrane being left behind, or by management of the patient on the side during the third stage. Hæmorrhage may occur and not be noted.

Symptoms.

Local Symptoms.

The bleeding may be internal or external or both. An enormous amount of blood may be dammed back, distending the vagina and uterus. When the hand is placed on the fundus, blood gushes out.

Sometimes there may be a terrific gush following delivery, but more often there is a steady oozing or a more copious flow. Sometimes the flow is interrupted, the uterus filling and emptying several times. The uterus is flabby and difficult to find, as opposed to the hard uterus when bleeding is from a laceration of the *cervix uteri* or vagina.

General Symptoms.

The general symptoms are well known and are those of blood loss; they vary in intensity with the rapidity of flow, the amount of blood loss and the condition of the patient. The usual symptoms are faintness, dizziness, air hunger, yawning, nausea, restlessness, thirst and as death approaches involuntary bowel movements and coma. Examination will show the face pallid, lips bluish-white and face cold and covered with beads of perspiration, the eyes sunken, respiration rapid and shallow, the pulse small and fast and finally absent from the wrist.

Prevention.

It must be admitted by all that the most effective preventive measure is the correct management of the third stage of labour. Over-stimulation of the uterus and premature attempts at expulsion of the placenta account for a large percentage of *post partum* hæmorrhages.

The patient is best treated on her back and the *fundus uteri* should be guarded by the left hand of the *accoucheur*. If it be necessary to leave the manipulation of the fundus (for example, for the resuscitation of the child) the fundus may be left alone and observed from time to time together with the general condition of the patient. It is better to dispense with manipulation of the fundus than to leave it in unskilled hands.

One of the most important measures of prevention is to wait at least half an hour before any attempt is made to expel the placenta and membranes. Various signs that the placenta is separated (elevation of level of fundus from below to above the umbilicus and lengthening of the cord) are known to us, but we have no such information about the separation of the membranes. Credé stressed this point very forcibly.

A complete understanding between the nurse and doctor is absolutely essential. This would appear easy to obtain, but very often they are working at cross purposes. Thus, it is advisable for a medical practitioner to restrict the number of nurses with whom he works. If the nurse and doctor are continually working together there will be better team work.

Prevention may be carried out by eliminating the previously mentioned causes. For example, this

may be done by refusing to attend women in their homes who are suffering from any toxæmia or who give a history of bleeding or of adherent placenta. It is almost criminal to allow a woman to go through labour in her home when she has any of the following conditions: *Placenta prævia*, fibromyomata, twins, hydramnios, debilitating diseases such as advanced pulmonary tuberculosis.

The best method of prevention is to see that the third stage of labour is properly conducted and above all to avoid over-stimulation of the *fundus uteri* and early attempts at expulsion of the placenta.

I should like to put forward a suggestion for lectures to be given on this subject to the various Branches of the British Medical Association and to obstetric nurses in the various suburban and country districts. The same lectures could be repeated every three or four years. Contempt of the seriousness of *post partum* hæmorrhage is formed very easily when an *accoucheur* has a series of normal confinements. To my mind the blame for a severe *post partum* hæmorrhage can be placed on the *accoucheur* in practically every instance.

Management.

The patient is treated on her back. The management of placental site hæmorrhage will depend on whether the placenta is retained in the uterus or not.

If the placenta is in the uterus and slight hæmorrhage occurs, gentle massage of the fundus will be found to control the bleeding in nearly every instance.

Should the hæmorrhage continue, a measure that has been described may be tried. This is suitable for women with lax abdominal muscles. The procedure is to lift the uterus higher into the abdominal cavity by exerting upward manual traction through the abdominal wall. It has been successful in my hands in one case.

If the hæmorrhage is severe, immediate steps must be taken to evacuate the uterus. First, an attempt at expulsion of the placenta is made by Credé's method. If this is not successful immediately, the placenta must be removed manually. Manual removal of the placenta in the home is a serious measure and there is a very definite risk of introducing infection. To lessen this risk of infection by urgent manual removal of the placenta, it is my plan to have two pairs of smooth rubber gloves sterilized for every confinement (one for the delivery of the child and one in readiness for immediate manual removal of the placenta). It is no more trouble to sterilize two pairs of gloves than one.

It is customary to use an intrauterine douche of hot saline solution for any manœuvre such as this. This is an excellent procedure in a hospital where one can be moderately sure of maintaining asepsis. But it is a very different proposition in most of the poorer homes of Sydney. Thus I have dispensed with an intrauterine douche as a routine measure after manual removal of the placenta and use it only in exceptional cases in the home, but use it as a routine in a properly equipped labour ward.

To aid uterine contraction and retraction, one cubic centimetre of "Pituitrin" or "Infundin" is given into the deltoid muscle and vigorous massage to the *fundus uteri* is carried out. In the vast majority of cases the bleeding is now controlled.

The Treatment of Post Partum Hæmorrhage After the Expulsion of the Placenta.

It is always necessary to make sure that the placenta and membranes are complete. It should be a routine practice to examine these carefully, always keeping in mind the possibility of the presence of a *placenta succenturiata*.

Slight hæmorrhage is effectively controlled by vigorous massage to the *fundus uteri*, with intramuscular injections of "Infundin" and aseptic ergot.

Severe hæmorrhage should be controlled by bimanual compression of the uterus, after making certain that the uterus is quite empty. No time should be wasted in uncertain methods of hæmostasis and bimanual compression of the uterus should be resorted to early and before the blood loss has become serious and the patient collapsed. The risk of carrying infection into the uterus is no greater than in giving a hot intrauterine douche and in packing the uterus, especially if there is a pair of sterilized rubber gloves in readiness for the emergency. Bimanual compression is the most effective method of controlling placental site hæmorrhage. It can be carried out for a considerable length of time without tiring the *accoucheur*, only a slight degree of pressure of the abdominal hand is necessary to control the bleeding.

Firm and strong pressure is tiring to the operator and also causes the patient a certain amount of shock. The method is to place the closed fist of the left hand in front of the *cervix uteri* and the fingers and palm of the right hand on the posterior surface of the fundus and body of the uterus. The amount of pressure necessary to secure hæmostasis should never produce shock, but should it occur it is best treated by a hypodermic injection of fifteen milligrammes (a quarter of a grain) of morphine. This should be given by the nurse while compression is being carried out. Bleeding is always controlled by bimanual compression and in practically every case the uterus becomes firmly contracted within a quarter of an hour.

Other methods of hæmostasis are plugging of the uterus and vagina (in my opinion inferior to bimanual compression), manual compression of the aorta (suitable in women with lax abdominal muscles), mechanical compression of the aorta by a Momburg's tourniquet.

The important thing is to have a plan of action in severe hæmorrhage.

When the placenta is *in utero*, empty the uterus by Credé's method or by manual removal of the placenta and then bimanual compression until the uterus is firm. Contraction and retraction are stimulated by injection of "Infundin" or a similar preparation.

After expulsion of the placenta, exploration of the uterus and bimanual compression of the uterus. These are the main features of a plan of action where there are no hospital facilities available.

After arrest of the hæmorrhage the following measures may be carried out. Warmth by hot bottles and blankets, elevation of the foot of the bed, the lower extremities may be firmly bandaged, subcutaneous injection of saline solution, brandy by mouth, coffee and brandy *per rectum*, morphine when shock is present. In extreme cases blood transfusion in the home could be carried out.

HYPERSENSITIVENESS TO LINSEED.

By CHARLES SUTHERLAND, M.B., B.S. (Melbourne), M.R.C.P. (London).
Honorary Physician to Asthma Clinic, Alfred Hospital, Melbourne.
(From the Baker Medical Research Institute.)

THE importance of hypersensitiveness to various substances is becoming widely recognized and in searching for it routine tests are commonly performed with epithelial products, such as horse dander, cat hair and feathers, with pollens and with certain powders, such as orris root and pyrethrum. But sometimes a patient who seems to be suffering definitely from hypersensitiveness to some substance, fails to respond to anything commonly used in testing the skin. Such patients usually are free of symptoms in certain localities and as a rule the nose shows typical vasomotor rhinitis with swollen pale mucous membrane.

It seems obvious that our list of substances which commonly excite symptoms, is far from complete yet and it is of the utmost importance that every effort should be made to detect other common offenders.

One patient was referred to me for investigation on account of rhinorrhœa which had persisted for over two years. As a rule she slept with a towel round her face to catch the clear watery discharge which poured from the nose and during the last eighteen months she had lost more than 12·7 kilograms (twenty-eight pounds) in weight. She was usually better if she went up country and examination revealed the typical "hay fever nose." No reactions were obtained to tests with over seventy pollens, forms of epithelium and foods except for small reactions to goose and duck feathers and house dust. Among subsequent tests a little crushed linseed was applied to a scratch with one-hundredth normal sodium hydroxide solution; in a few minutes a violent urticarial reaction spread up and down the arm and there was intense itching. The patient then stated that for the past two years she had used linseed oil and kerosene as a floor polish. Of course she was told to eliminate linseed as far as possible from the household and during the next few weeks there was some improvement, but as symptoms were still very troublesome it was decided to desensitize her. An extract was prepared at the Commonwealth Serum Laboratories and for comparison a drop of it was applied to a scratch on one forearm and the crude linseed with centinormal sodium hydroxide solution was applied to the other arm. Both gave about equally large reactions and precipitated a brisk attack of rhinorrhœa, but luckily no other unpleasant symptoms appeared. Serious constitutional upsets have followed intradermal tests with various substances, but it is most unusual to get any trouble with scratch tests. Desensitization has been tedious as even 0·01 cubic centimetre of a one in one thousand dilution of the original extract produced much swelling when injected subcutaneously. She can now tolerate, after four months' treatment, 0·4 cubic centimetre of a 1:10 dilution and practically no local reaction ensues. There have been no nasal symptoms for two months.

This case indicates the danger of incomplete testing as the reactions to feathers might have led to the view that they were the chief factor and desensitization to them would almost certainly have been futile.

Another patient who had had asthma for twenty years, showed good reactions to duck feathers and house dust and was strikingly improved by desensitization. However, he was suddenly attacked again with violent asthma and this persisted for a fortnight and was especially bad when he was at home. He had been tested at a time when I had not realized the importance of linseed and it had not been tried, but it was now found to give a good reaction. When asked if he had been using linseed he stated that the painters had been working in his house for a fortnight. It seems likely that this accounted for his attacks.

A number of similar cases have been detected and there is no doubt that linseed is an important cause of symptoms in Australia. It is mentioned occasionally in the literature, but its importance does not appear to have been sufficiently emphasized. As a rule symptoms are shown in the respiratory tract, but I have seen at least one case of dermatitis apparently due to idiosyncrasy to it. Hope, Hanna and Stallybrass[1] mention that workers with linseed frequently develope an acute eczematous condition of the face, arms and back of the legs. Calcutta linseed appears to be the only variety that causes it. It is not known whether it is the linseed or some parasite associated with it which is responsible. Workers in skutching mills (where flax is handled) are prone to ophthalmia, asthma and pulmonary tuberculosis. Boys working on the "heckling" machines with flax are exposed to much organic dust and those exposed for the first time are prone to have "mill fever." This is a febrile reaction lasting three or four days and apparently anaphylactic in nature. Increasing dyspnœa and cough develope in some and typical asthma ensues, with bronchial spasm and turgescence of the bronchial mucosa.

It is interesting to note that some patients give equally large reactions to the crude linseed and to the liquid extract. Others give a large reaction to the crude material and no reaction to the extract. Even intradermal injections of the extract give only faint reactions, so it seems that there are at least two substances in the seed capable of exciting reactions.

Some individuals fail to give reactions to any forms of epithelium, but give good reactions to house dust. So far all efforts to identify the active principle in the dust have been unsuccessful, but it has been definitely shown that it is not derived from any of the common forms of epithelium. My experience indicates that linseed is not a factor, as many persons who react to house dust, do not react at all to linseed. On the other hand, all persons so far giving a positive reaction to linseed, react in some degree to house dust.

Summary.

Routine investigation of allergic patients has shown that linseed (flax seed) is frequently a cause of symptoms.

Reference.

(1) Hope, Hanna and Stallybrass: "Industrial Hygiene and Medicine," London, 1923.

Reports of Cases.

AN UNUSUAL STRAIN OF EBERTHELLA TYPHI (BACILLUS TYPHOSUS), ISOLATED AT TOWNSVILLE, NORTH QUEENSLAND.[1]

By A. B. LILLEY, M.B., Ch.M. (Sydney),
Bacteriologist, Royal Prince Alfred Hospital, Sydney.

THE organism to be described was isolated while I was attached to the staff of the Australian Institute of Tropical Medicine, Townsville, Queensland. I am indebted to Dr. H. M. Hollingworth, Superintendent of the Townsville General Hospital, for supplying the clinical notes.

Clinical History.

A patient, K.G., a male, *ætatis* sixty years, was admitted to the Townsville General Hospital on November 2, 1927, complaining of a swelling on the left side of the face which he had noticed to be present for the past six months.

On examination there was seen to be a tumour approximately 2·5 by 1·25 centimetres (one inch by one half inch) over the left lower jaw, adherent to the mandible and hard to the touch.

The Wassermann test yielded no reaction.

At operation the tumour was removed together with one-half of the mandible (left) submaxillary lymph and salivary glands and the submental lymph glands. Microscopical examination of the excised tissue revealed extensive infiltration by squamous epithelial cells with much cell nest formation and inflammatory reaction. The appearance was that of a squamous epithelioma.

Some little time after operation the wound broke down and a sinus formed. On February 8, 1928, a further operation was performed on the sinus.

On February 14, 1928, the patient complained of feverishness, headache, pain in the body and limbs.

[1]Read at a meeting of the New South Wales Branch of the British Medical Association on June 14, 1928.

Two days later he vomited a few times. The temperature and pulse chart is shown herewith.

On February 17 the pains were easier, but he still complained of nausea.

On February 19 he had severe abdominal pain and vomiting of a watery brown fluid, anorexia and sleeplessness. He was tender and somewhat rigid over the gall bladder area.

On February 21 blood culture was performed into 0·5% sodium taurocholate broth and serum was taken for the performance of the Widal test.

The Widal test gave no reaction for *Eberthella typhi* (*Bacillus typhosus*), *Salmonella paratyphi* (*Bacillus paratyphosus A.*) and *Salmonella schottmülleri* (*Bacillus paratyphosus B.*) in dilutions of 1 in 20 to 1 to 200. On February 22 the blood culture was positive. A subculture was made into broth and a MacConkey plate was inoculated.

After six hours' incubation the organism in the broth culture was non-motile and on staining proved to be a Gram-negative bacillus in pure culture.

On February 23 the MacConkey plate showed a pure culture of a non-lactose fermenting Gram-negative coliform bacillus. A colony was picked off from this plate and inoculated into sugars and broth for a further motility test and to an agar slope for agglutination test.

On February 24 the organism was still non-motile after culture for six hours in broth.

The sugar reactions after twenty-four hours' incubation were as follows: Acid without gas was produced in glucose, maltose and mannite. There was no change in dulcite, saccharose and lactose (reaction still alkaline). The organism isolated from the patient's blood was accordingly on this day tested against specific agglutinating sera as shown in Table I.

TABLE I.

Agglutinating Serum.	Result.
Dysentery (Flexner) ..	No agglutination 1 in 25 to 1 in 250.
Typhoid	Agglutination +++ to 1 in 250 (not taken further).
Patient (homologous) ..	Agglutination +++ to 1 in 250 (not taken further).

The Widal test of the patient's serum was repeated on this day as shown in Table II.

TABLE II.

Bacterial Emulsion.	Dilutions of Patient's Serum.			
	1 in 25	50	125	250
B. typhosus (Rawlings)	Positive ++	No agglutination	No agglutination	No agglutination

On February 25 the agglutination reactions which had on February 24 been completed as far as dilutions of 1 in 250, were extended as shown in Table III.

TABLE III.

Bacterial Emulsion.	*B. typhosus* Anti-Serum (C.S.L.) Titre 4,000.			
	1 in 1,000	2,000	3,000	4,000
Organism isolated from blood of patient	Positive +++	Positive +++	Positive ++	Positive +
B. typhosus (Rawlings), the strain used for the Widal test	Positive +++	Positive +++	Positive +++	Positive +++

Since on February 24 it had been shown that the patient's serum possessed agglutinins to its homologous

organism, but was as yet incapable of agglutinating an emulsion of *Bacillus typhosus* (as evidenced by the absence of response to the Widal test), it was obviously essential to prove that the emulsion being used as a typhoid emulsion for the performance of the Widal test was actually a typhoid emulsion. This was proved as shown in the above test by putting it up against a typhoid agglutinating serum (which serum agglutinated it to full titre).

No non-lactose-fermenting organisms were grown after twenty-four hours' incubation from fæces and urine which had been plated out on MacConkey medium on February 24.

On February 26 the agglutination reaction of the patient's serum against its homologous organism (complete agglutination had been obtained as far as a dilution of 1 in 250 on February 24) was extended with the same specimen of serum with the result shown in Table IV.

TABLE IV.

Bacterial Emulsion.	Serum of Patient.				
	Dilution of 1 in 500	1,000	1,250	1,700	2,500
Organism isolated from patient's blood	Positive +++	Positive +++	Positive +++	Positive ++	Positive +

On February 29 the Widal test was repeated for the third time and again no agglutination occurred for *Eberthella typhi*, *Salmonella paratyphi* and *Salmonella schottmülleri* in dilutions of 1 in 25 to 1 in 250.

Further cultural reactions of the organism were carried out in the next few days and the following results were obtained: Gelatine, not liquefied; milk, slight transient acidity returning to alkalinity; indol, not produced; nitrates, reduced to nitrites; sugars, produced acid without the formation of gas in lævulose, sorbite, galactose, raffinose, mannite, dextrin, maltose and glucose; motility, after the organism had been on culture for about fourteen days it developed feeble motility when subcultured into broth and examined after six hours' incubation.

Summary and Discussion.

1. Motility and specificity of antibody production are such well marked characteristics of *Eberthella typhi* that any departure from these characteristics must excite interest. In the present instance there has been isolated from human blood in a case of continued fever an organism which gives the cultural characteristics of *Eberthella typhi* and which is agglutinated to high titre by a typhoid agglutinating serum, but which is non-motile. Non-motile strains of *Eberthella typhi* (*Bacillus typhosus*) have been described previously.[1] [2]

2. The organism had stimulated in the body of the patient the production of antibodies (agglutinins) to a high degree, since the patient's serum was capable of agglutinating its homologous organisms as far as a dilution of 1 in 2,500.

It was to be expected therefore that the patient's serum would prove just as capable of agglutinating an emulsion of the stock strain of *Eberthella typhi* (Rawlings), that is, give a Widal reaction. When I performed this test, however, I found that the patient's serum would not agglutinate *Eberthella typhi* (Rawlings), even in a dilution of 1 in 25. The performance of this test on three separate occasions on different specimens of serum still failed to produce a positive result, except in the one instance on February 24, 1928, when there was partial agglutination in a dilution of 1 in 25.

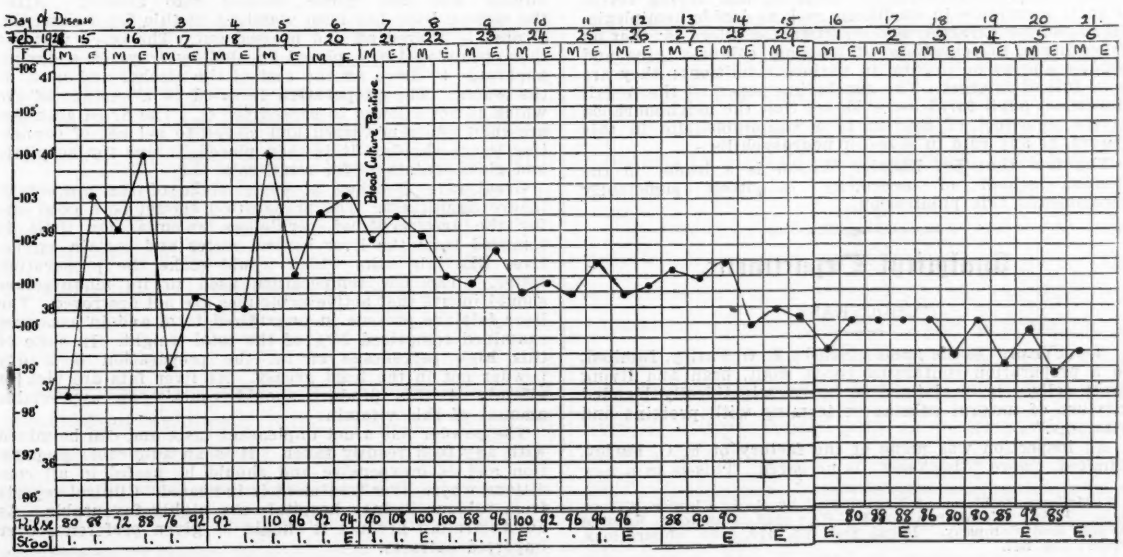
I am forced to the conclusion, therefore, that here is an organism which is undoubtedly *Eberthella typhi*, but which is capable of producing an agglutinin which fails to agglutinate a stock strain of *Eberthella typhi* (Rawlings). On the other hand the agglutinin produced by a stock strain of *Eberthella typhi* (Rawlings) is capable of agglutinating the organism described here.

The other striking feature of the organism is its lack of motility. In this regard the patient did not give any history of having had typhoid, paratyphoid *A* and *B* inoculation, as was the case in the organism described by Archibald.[3] The case is of interest also because of the fact that at the time the patient contracted the illness, he had been an in-patient in the hospital for a period of over three months. No patient with typhoid fever was present in the hospital at the time, nor had there been one for some considerable time preceding the onset of his illness.

It is obvious that the performance of an agglutinin absorption test should have been done and it is unfortunate that the opportunity to do so did not occur.

References.

[1] J. A. Arkwright: "Importance of Motility of Bacteria in Classification and Diagnosis with Special Reference to B. Pseudo-Tuberculosis Rodentium," *The Lancet*, January 1, 1927, page 13.



(20) R. G. Archibald: "An Unusual Case of Infection with a Non-Motile Strain of B. Typhosus in an Individual Previously Inoculated with T.A.B. Vaccine," *Journal of Tropical Medicine and Hygiene*, May 16, 1927.

Reviews.

NEUROLOGICAL ESSAYS.

MODERN problems in neurology is the title given by Dr. Kinnier Wilson to a volume composed of a series of papers he has published during the past few years in various journals.[1] The list comprises the epilepsies, the narcolepsies, striatal disease, pathological laughing and crying, dysæsthesiæ and their interpretation and the Argyll Robertson pupil. Of these the chief is undoubtedly that on striatal motor function. The study arose from his original investigation of progressive lenticular degeneration (Wilson's disease) and formed the subject of his Croonian lectures delivered before the Royal College of Physicians of London. The subject is most elaborately and critically discussed. Disorders of motility and muscle tone arising in progressive lenticular degeneration, Parkinson's disease, Parkinsonism, chorea and so on are viewed from every possible angle. It is made plain first that the symptom-complex which has been erected on the ganglion, known as the *corpus striatum*, is hopelessly topheavy and must be rigorously stripped of non-essentials and secondly that in the attempt to assign separate functions to the composite parts of the whole ganglion speculation has widely outrun clinical and pathological facts. Allowing for many gaps in our knowledge of striatal disease remaining to be filled, the conclusion emerges that: "the cardinal clinical features of disease of the striate body may be summed up in three words—variability in muscle tonus, the appearance of involuntary movements and the absence of paralysis in the strict sense of the term."

In the study of epilepsy the reader will find not so much a clinical description of the disorder as a reasoned discussion of what epilepsy is, and he will inevitably agree that it is more correct clinically and physiologically to speak of "the epilepsies" than of "epilepsy." The chapter on epileptic variants, on the other hand, is descriptive and gives a useful clinical account of those attacks, sometimes called equivalents or substitution phenomena related to and yet widely different from the phenomena of epilepsy as usually understood. The account of the narcolepsies is also mainly clinical and we think many familiar with these attacks will agree with the writer's thought that they present compelling analogies to the epilepsies.

The paper on pathological laughing and crying refers to their occurrence in conditions such as double hemiplegia, pseudo-bulbar paralysis and disseminated sclerosis; that on dysæsthesiæ to sensations of burning, coldness, pins and needles and so on arising in various conditions; they are both full of interest. The concluding paper on the Argyll Robertson pupil furnishes evidence that the neighbourhood of the aqueduct of Sylvius is a vulnerable site in this regard, at any rate in cases of neurosyphilis.

The fact that Dr. Kinnier Wilson is a leader in the London school of neurology in itself sufficiently recommends this publication.

Analytical Department.

"CHOCYEAST."

"CHOCYEAST" is the name given by E. O. Farley, Limited, to a preparation containing cocoa, malt, bran and living yeast cells. It is claimed to be "a scientifically blended mixture of natural substances teeming with proteins and vitamins."

An inspection was made of the factory of E. O. Farley, Limited, where "ChocYeast" is prepared. This is in a new building and is kept scrupulously clean. Washed yeast, obtained in brick form, is mixed with very finely powdered malt and water to form a stiff paste. This is rolled out into thin slabs which are broken into small pieces and dried at room temperature in a current of air. When dry, the cake is ground up and mixed in a machine with powdered, somewhat defatted cocoa and finely powdered bran. For the malt, whole malt is ground to a very fine powder in a completely enclosed machine. The bran is roasted at a low temperature and ground to a fine powder. The preparation is then placed in paper-lined tinplate containers. The preparation throughout was carried out in a careful and cleanly manner.

Analysis.

Samples of "ChocYeast" were obtained at the factory and purchased on the open market.

	I	II
Water	8·42%	9·36%
Ash	2·75%	2·83%
Available carbohydrate	52·57%	52·09%
Protein	16·84%	16·46%
Fat	7·70%	7·04%
Crude fibre	11·72%	12·22%

The agreement between the two samples is reasonably good taking into consideration the nature of the mixture.

The preparation is of a light chocolate colour with a pleasant smell and taste. Living yeasts were abundant in the preparation, but no attempt was made to count them.

For use "ChocYeast" is mixed with warm milk and drunk like cocoa. Prepared in this way it makes a very palatable drink and one rich in food materials. It is claimed that the yeast and the bran give the mixture a laxative effect. If it is desired to give living yeast cells for this or other purpose "ChocYeast" offers a very pleasant and convenient way of so doing. It is a good, agreeable and nourishing preparation.

"KARNA VITA" DESICCATED LIVER.

"KARNA VITA" is the name given to a preparation of desiccated liver made by the Karna Vita Company at the State Abattoirs, Homebush Bay. It is claimed that it can usefully be employed instead of liver extracts in the treatment of pernicious anæmia and other anæmias.

An inspection was made of the process of manufacture at Homebush. Carefully selected ox livers are placed in cold storage for twenty-four hours. They are then finely minced and the mince thrown into alcohol. After the minced liver has been standing in this for a time, the alcohol is filtered off and pressed out. The thick paste is then granulated and placed in shallow trays in a drying apparatus to drive off the rest of the alcohol and most of the water. The preparation is dried in a current of air which is not allowed to exceed 40° C.. The dried granules are then finely powdered and sieved to get rid of connective tissue shreds. It is then placed in tins for hospitals and glass containers for retail sale.

Great care is taken at every stage of the process to insure cleanliness. The preparation should be a good one for the treatment of pernicious anæmia, for little is removed from the liver beyond water and certain extractives like bile salts which would make the preparation bitter. The low temperature used in its manufacture should insure that active principles are not destroyed. The liver fats are present in unoxidized form and in a sample examined comprised 26% of the total weight. In spite of this high percentage of fat the preparation is a dry powder not in the least greasy. As liver fats are rich in vitamin *A* this product should contain an appreciable amount of this vitamin.

The powder has a not unpleasant taste and can be mixed with any food readily taken. It is an admirable preparation and is inexpensive and should be useful in all conditions where liver treatment is indicated. Clinical reports from various Sydney hospitals are highly favourable. As an Australian product it should be given preference over imported extracts.

[1] "Modern Problems of Neurology," by S. A. Kinnier Wilson, M.D., B.Sc. (Edinburgh), F.R.C.P.; 1928. London: Edward Arnold and Company. Royal 8vo., pp. 370, with illustrations. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, MAY 18, 1929.

The Third Session of Congress.

IT is a platitude to call attention to the value of meetings of scientific societies and of scientists for the purpose of discussion. The congress idea, an innovation of last century, has become firmly rooted in all cultured countries and its application has increased rapidly, as scientific thought and higher education have become more universal. Cultural progress is largely dependent on challenges to authority. The personality of the challenger counts for much when the new doctrines savour of revolution or when fundamental data are incapable of definite proof. Every scientist recognizes that there is more to be learned in listening to the reading of a paper and in participating in a debate than in the leisurely reading of a printed article. Points that appear to be obscure or ambiguous in a paper, can be explained and elucidated when attention is called to lack of clarity. It is unnecessary to labour this thesis, for searchers after truth have long since admitted the immense value of congresses.

The term congress is somewhat wide. In Europe international scientific congresses are gatherings of great size at which communications are presented by leaders in the various branches of scientific endeavour. The papers, as a rule, are prepared with extreme care and prolonged consideration. The world regards these meetings as important and in consequence every *rapporteur* recognizes his responsibility and endeavours to make his contribution worthy of a great occasion. Then there are national congresses. In the past some of the national congresses have aimed at and attained a standard of excellence equal or nearly equal to that of an international congress. Usually, however, the national gatherings obtain their character from the fact that many of the attending members are personally known to each other. The discussions tend to become somewhat less formal and the demands placed on the readers of the chief papers are perhaps less exacting than they are in a larger forum. The third kind of congress is the annual meeting of a single organization. At times the members take their scientific functions seriously, especially if the body is little homogeneous, as is the case with the British and the Australasian Associations for the Advancement of Science. Often, however, the annual meetings of scientific organizations are social picnics relieved by a few scientific meetings. There is often a lack of enthusiasm both in the preparation for the debates and in their conduct. It is noted that the organizers are glad to accept almost any paper that is offered and the inevitable result is that the annual meeting of many of our scientific societies does not rank very high among the congresses.

The third session of the Australasian Medical Congress (British Medical Association) will be held in Sydney in about four months. It rests with the members of the British Medical Association in the several States of Australia and in New Zealand whether it will bear the character of an international, a national or a provincial congress. The experience of the past heartens us to urge the members to aim at the highest plane. Australasia comprises a very large section of the world and for this reason there is little danger of the session becoming a family reunion. We have many earnest workers who have important messages to give, many who are in a position to contribute to the progress of medical knowledge, many who are bold enough to emancipate themselves from authority when evidence is available that accepted doctrines are insecurely based.

We learn with regret that relatively few have enrolled as members up to the present date. The time is short and it is essential that the Executive Committee should have reasonable opportunity of making the preparations for the meetings. Those who are still in doubt whether or not they will be able to attend, should join as members now and make a real effort to be in Sydney on September 2, 1929. In the next place, if the session is to be a success, such as we believe it will be, every member who would like to read a paper, should be far

advanced with the work of preparation and should offer his contribution to the secretaries of the sections without delay. On another page in this issue we publish again a summary of the rules governing the offering and reading of papers. It may be wise to amplify these notes in one or two directions. Brevity consistent with lucidity should be aimed at. Nothing is more tedious than a long, rambling paper; in addition a long paper robs the readers of other papers and those who desire to take part in discussions, of opportunities. We understand that the time limits will be imposed with strictness. A contribution to an important congress should bear a definite message. A summary of work done by others, a reedition of an author's previous work without essential additions, a recital of suggestions or hypotheses without secure foundation should have no place on the programme. Our appeal to the medical profession in Australasia is to join the third session of Congress without delay and to those who join, to regard it as a personal responsibility to give this meeting the character of an international gathering. The organizers of the session are doing their work in a most admirable manner.

Current Comment.

SMALL POX AND VACCINATION.

THE adequacy of measures directed towards the control of small pox is a matter of grave concern to Australia. The Commonwealth enjoys a great variety of climate and a large area in the north lies within the tropical zone; several of its larger ports are but a few days' journey from countries in which small pox is endemic. In regard to vaccination, as with many other matters of medical importance, there is no uniformity among the States. In New South Wales there is no statutory provision for compulsory vaccination. Public vaccinators have, however, been appointed. The effectiveness of this arrangement may be judged from the fact that during 1919 of 55,740 children medically examined by the Education Department 17% had been vaccinated. In Victoria compulsory vaccination is enforced, but this enforcement is subject to a "conscience" clause. From the year 1873 to 1918 it is estimated that 72% of the children whose births were registered, were vaccinated. During 1926 1,318 children were vaccinated; this is less than 4% of the children whose births were registered. Although in Queensland there is provision for compulsory vaccination, the operation of the act has not been proclaimed. According to the *Vaccination Act* of 1882 vaccination in South Australia was compulsory, but in 1917 an act to suspend compulsory vaccination was passed. Only two vaccinations were reported in this State in 1926. In Western Australia vaccination was compulsory under the *Vaccination Act*, 1878; with the passage in 1911 of the *Health Act* which contained a "conscientious objection" clause, the first act lost most of its usefulness. In Tasmania all infants are required under the *Vaccination Act*, 1898, to be vaccinated before the age of twelve months unless either a statutory declaration of conscientious objection is made or a medical certificate of unfitness is received.

According to the Commonwealth *Quarantine Act*, 1908, which is administered by the Minister for Trade and Customs of the Commonwealth Government, "a quarantine officer may in the case of small pox require any person subject to quarantine or performing quarantine to be vaccinated and any person so required to be vaccinated shall submit to be vaccinated accordingly." The penalty attached to this section is £5. The act further provides that: "A quarantine officer shall not require any person to be vaccinated unless in his opinion vaccination is necessary for the protection of persons subject to quarantine or performing quarantine or for the prevention of the spread of the disease of small pox."

In view of this state of affairs it will serve a useful purpose to consider a discussion[1] which took place early this year before the Royal Society of Medicine, on vaccination against small pox "in the light of recent experience." The very title of the discussion is suggestive that recent experience calls for consideration of the whole question. J. C. G. Ledingham opened the discussion. After dealing with the question of the results of the passage of virus through animals on the lesions which the virus would produce, he turned to the different types of small pox. He referred to the work of Armstrong and Cleland and Ferguson in Australia and to the scarcity of information on alastrim as it occurs in England. Alastrim in his opinion is a small pox variant. It was this condition which broke out in New South Wales in June, 1913. Ledingham's summary of the experimental evidence in support of the thesis that alastrim is a variant of small pox is interesting. It is readily communicable to the monkey by scarification. In the rabbit alastrim virus produces typical vaccinia-like vesicles only after repeated passage, but when inoculated intradermally in the rabit, material direct from man produces a characteristic reaction which is neutralizable by antivaccinia serum. On the rabbit's cornea the virus usually gives a positive response to Paul's test and Guarnieri bodies may be found. Paschen's elementary bodies may be found in smears from early vesicles and a precipitin reaction has been obtained with alastrim material and antivaccinia serum. Ledingham regards alastrim as a stable variant of small pox and he states that when it has been alleged that the case mortality of

[1] *Proceedings of the Royal Society of Medicine*, February, 1929.

alastrim has risen to a significant percentage in a country in which it has previously been well under unity in spite of thousands of cases, it will be found that sufficient consideration has not been given to the possibility that the virulent form of small pox has been somewhere incident alongside the alastrim, but without definite transitions. Another aspect of "recent experience" mentioned by Ledingham was that of post-vaccinal encephalitis. This lesion which is fortunately rare, occurs as a rule in children of school age who have been vaccinated for the first time. Ledingham draws attention to the remarkable constancy of the interval of time which elapses between the vaccination and the appearance of nervous symptoms. While much is made of this by those who regard vaccination as responsible for post-vaccinal encephalitis, he holds that the constancy of this period may fit equally well with a view that presupposes some latent neurotropic virus in the vaccinated persons to be activated by the vaccinia.

These are the main considerations of modern experience on which the subsequent discussion was based. The participants apparently wished to determine whether in view of the relatively mild nature of the alastrim seen in Great Britain, some modification of compulsory vaccination might be recommended. It is impossible in the space at disposal to give a *résumé* of all the views expressed. Opinions were divided. Only one member was in favour of compulsory vaccination. Others thought that in the event of an outbreak of virulent small pox the vaccination defences of the country could be mobilized quickly and with good effect. Others were frankly of the opinion that alastrim was such a mild infection that it was not worth preventing. Ledingham thought that the dose at vaccination might be reduced to one insertion and that the deficiency of this dosage might be made up by subsequent revaccination. He laid great stress on the education of the public by "informative and authoritative pamphlets and the help of an intelligent press."

Before a decision is arrived at in connexion with this matter at least two questions have to be investigated. In the first place it must be known why in many countries there is freedom from the disease and why this "stable variant," alastrim, has in many areas taken the place of the erstwhile virulent variola. In the second place it is important to discover the reason for the appearance of post-vaccinal encephalitis, for this more than anything else will influence the popular mind against vaccination. The first question is as difficult to decide as the last. In considering it the following must be taken into account: the influence of herd immunity, the influence of vaccination on the community as a whole, the effect of quarantine and the nature of the infecting agent. Several facts may be stated: Vaccination and revaccination can and do protect the individual against variola; vaccination and efficient quarantine can keep an uninfected community from becoming infected; the virulent form of small pox may exist in a community by the side of alastrim without any definite transitions being apparent. Herd immunity may be described as the immunity which results from unrecognized or mild infections scattered throughout a community. The work of Dudley has shown that herd immunity is a real factor in the epidemiology of such diseases as diphtheria and scarlet fever. That it plays a part in the epidemiology of small pox need not be questioned. Herd immunity will not result in a community all of whose members are not susceptible. Compulsory vaccination and revaccination will do this. These procedures were carried out in Germany before the war with signal success. Methods of compulsion were used when objections were raised and the result was a community immune to small pox. Any infected persons who got past the quarantine barrier, found themselves in a community in which they could not do much harm. As far as the infecting agent is concerned, it must be remembered that though alastrim is the dominant form of small pox in many countries today, virulent small pox may arise at any moment, as virulent influenza did a decade ago. Before the cry is raised that Australians would not submit to compulsory vaccination, it should be pointed out that no proper attempt has been made to move in this direction. All the States except New South Wales have or have had statutory provisions for compulsion. In some no real attempt has been made to enforce the act and in the majority the "conscientious objection" clauses are too readily applicable. Education of the general public should certainly be carried out, but this should be an additional weapon in a wholehearted attempt to introduce compulsory vaccination.

This leads to the second question previously referred to. Settlement of this will make compulsory vaccination more acceptable. Ledingham's view has already been mentioned. It must again be emphasized that post-vaccinal encephalitis occurs most often at school age. If the encephalitic changes were due to the vaccine, it would be difficult to explain this frequency. On the evidence available it seems much more likely that the involvement of the central nervous system depends on some factors present in the patient or his environment. It is in this direction that research must be extended.

THE BRITISH JOURNAL OF UROLOGY.

BRITISH urologists have for some years laboured under the disadvantage of having no journal exclusively devoted to their interests. *The British Journal of Urology* which has just appeared, will fill a definite need and we heartily welcome its appearance. The first number augurs well for future issues. The editors, Dr. Frank Kidd and Dr. Winsbury White, are to be congratulated on its appearance and general make-up. The journal is printed by Constable and Company, of 10 and 12, Orange Street, London, W.C.2. Dr. R. Gordon Craig represents Australia on the editorial committee and Dr. R. Campbell Begg represents New Zealand.

Abstracts from Current Medical Literature.

BACTERIOLOGY AND IMMUNOLOGY.

Bacteriology of the Gall Bladder.

K. L. PESCH AND V. HOFFMANN (*Münchener Medizinische Wochenschrift*, October 5, 1928) have investigated fifty cases of gall bladder infection to determine the presence of bacteria and their effect on the causation of cholecystitis and cholelithiasis. They disagree with other authors who state that bacteria, especially the staphylococcus, may be cultivated from practically every affected gall bladder. From their work they conclude that many gall bladders are sterile and that in infected gall bladders the predominating organism is the *Bacillus coli*. They draw attention to the difficulty of preventing accidental contamination of the cultures. This was obviated in their cases, first, by sterilizing the culture tubes with the surgical dressings and secondly, by incubating the tubes for three days before opening them to obtain specimens. Usually cultures are taken every twenty-four hours and there is a great risk of accidental aerial infection with staphylococci in particular. They obtained sterile cultures from 24% of the gall bladders in which histological examination revealed bacterial inflammatory changes. In two cases of fatal staphylococcal septicæmia no organisms were found in the gall bladder, nor was there any inflammatory reaction. They consider that staphylococcal infection plays a very small part in biliary infections and if these organisms are found in a series of cases, care must be taken to exclude them as a purely accidental infection.

Active Immunization Against Diphtheria.

W. STOELTZNER (*Deutsche Medizinische Wochenschrift*, January 4, 1929) refers to the increased virulence of diphtheria which has been noted not only in Germany, but also elsewhere throughout the world. The clinical type of the disease has altered and many more severe toxic forms have been noted. Death occurs in the first or second week from vaso-motor or cardiac paralysis and it is noteworthy that the malignant form is more liable to attack older children. Even enormous doses of serum have had little effect in these infections. One reason advanced for this increased mortality has been the prevalence of mixed infections, notably streptococcal. Against this is the fact that streptococci are often found in association with diphtheria bacilli in non-malignant infections. They are also not present in at least half of the severe infections. Death is caused by severe diphtheritic infection which does not resemble a streptococcal sepsis. The author is of the opinion that in epidemics which cannot be controlled with serum, little faith can be placed in the value of immunization to prevent the occurrence of diphtheria. Despite extensive immunization in America the incidence of diphtheria has not decreased, while the mortality has risen during recent years. Although he takes a pessimistic view of the value of immunization to prevent the severe toxic infections, the author considers that it is of great value in protecting against the milder forms. Finally, he warns the medical profession to make it quite clear to the public that such immunization is not a permanent protection against diphtheria.

Direct Culture of Koch's Bacillus from the Sputum.

IN the sputum of a great number of patients who present symptoms suggestive of active pulmonary tuberculosis, tubercle bacilli are not found after repeated microscopical examinations. Inoculation of guinea-pigs is a long, difficult and expensive process when many patients are to be examined. The discovery of the bacillus in the sputum is regarded as such an important point that J. C. Suque (*Boletin Tecnico de la Direccion General de Sanidad*, January, 1929) investigated various methods of direct cultivation of the bacilli from the sputum. His earliest attempts were failures owing to the contamination of even the most carefully collected samples. Other methods required an inconveniently large quantity of sputum. A modification of the method used by Hohn gave him great satisfaction. The culture medium consists of fresh eggs and acid glycerinated broth, the latter made from meat extract, peptone and sodium chloride. He gives a detailed description of the technique of the preparation of the medium. By this method he has obtained a 20% positive result in sputa in which previously no tubercle bacilli could be found. Roloff, working among sanatorium patients in Germany, obtained results approximately equivalent to those obtained by Suque.

Yellow Fever.

N. PAUL HUDSON, JOHANNES H. BAUER AND CORNELIUS B. PHILIP (*American Journal of Tropical Medicine*, January, 1929) record the results of their observations in Africa on the protective qualities of sera of persons recovered from West African and American yellow fever. Experiments were made with monkeys (*Macacus rhesus*), the virus used being blood of a monkey suffering from experimentally produced yellow fever on the first day of the disease. The American sera were obtained and packed under tropical conditions and carried to Africa in a ship's refrigerator. Among the African sera was a sample consisting of a mixture of sera from several African natives taken within one month after their illness. This pooled serum produced immunity in all the experiments save one, in which only 0·1 cubic centimetre was injected subcutaneously. Protection was given in a lower percentage when a subcutaneous injection of serum was made simultaneously with a subcutaneous injection of virus than when the serum was administered intraperitoneally one hour before the injection of virus. Four of eleven American sera and all the African sera conferred immunity against African yellow fever, though not in every monkey inoculated. Several monkeys had attacks of pyrexia and appeared to suffer from yellow fever in a mild form. All control monkeys died of yellow fever. The authors suggest that the failure of some of the American sera to give protection may have been due to deterioration in transit. The evidence regarding the duration of immunity after an attack of yellow fever was inconclusive. One American serum taken from a person about seven years after an attack conferred immunity, while some from later infections failed.

Romanowsky Stains in the Tropics.

J. L. PAWAN (*Annals of Tropical Medicine and Parasitology*, November 9, 1928) describes his method of storing Romanowsky stains (Giemsha, Leishman and Wright). He states that the rapid deterioration of these stains under tropical conditions is due to alkalinity. Most glasses are composed of soda glass which rapidly corrodes, producing a thin film of surface alkali which is readily taken up by distilled water. This change does not take place to so great an extent with "Pyrex" glass, the alkaline content of which is 4·6% as compared with 12% to 18% in ordinary glass. Pawan makes a fresh supply of distilled water every one to five weeks and keeps his stains for as long as six or nine months, merely adding an occasional small amount of methyl alcohol to replace that lost by evaporation.

Yeast-like Organisms of Human Origin.

MARGARET J. KENNEDY (*Journal of Infectious Diseases*, January, 1929) has investigated the yeast-like organisms which are found either in pure culture or in conjunction with bacteria in the routine bacteriological examinations in any large hospital. A group of these organisms was examined to determine their importance, if any, as an ætiological factor in disease. Twelve of these yeast-like organisms were obtained from sputum, throat, stool and pus cultures and were studied in regard to their virulence, cultural and serological reactions. Eleven of the twelve were virulent to some degree for laboratory animals. The degree of pathogenicity varied from production of inflammation and abscesses to death. One strain was avirulent for both guinea-pigs and rabbits, although it had been repeatedly isolated from the stools of a patient with chronic colitis. Animal

immunization produced serum high in agglutinins and complement fixation antibodies. Agglutination and complement fixation indicated a great difference between the commercial yeast and the *monilia* isolated from human sources, although one strain was closely allied to the commercial yeast. In the cultures the yeast-like organisms isolated were *monilia*, producing mycelium and having no ascospores. Identification of the yeasts according to the table given by Castellani was difficult, since one or more cultural reactions usually differed from those given in his classification. In the small group studied there was no clear-cut correlation between source, cultural and serological reactions.

Upper Respiratory and Middle Ear Infections.

JOHN H. FISHER (*The Journal of Infectious Diseases*, January, 1929) has reported the results of experiments on animals with organisms isolated from acute *otitis media* and mastoiditis in human beings. Forty-six human middle ear infections were studied bacteriologically and in fifteen of these the pathogenicity of the isolated bacteria was tested by intranasal inoculations of rabbits. The *Streptococcus hæmolyticus* was the most frequently found exciting organism while staphylococcus and pneumococcus were next in frequency. Of the fifteen strains used eleven were *Streptococcus hæmolyticus* (*pyogenes*), two were *Streptococcus hæmolyticus* (*infrequens*) and two pneumococcus Type II. Before the intranasal inoculations were given, the nasal passages of the rabbits were swabbed with a 50% aqueous solution of phenol. Eleven of these rabbits died and in each instance acute purulent paranasal sinusitis and acute *otitis media* were found. Acute pleuritis and empyema, lobular pneumonia, lung abscesses, acute pericarditis and septicæmia were found as associated lesions in some of the rabbits. From all these lesions organisms similar to those inoculated intranasally were recovered. The experiments were controlled by two groups of rabbits, one group receiving culture only intranasally, the other receiving phenol swabbing only and no culture. None of the rabbits in these two groups died. Some factor which lowers the resistance of the nasal mucosa is necessary for the production of upper respiratory and middle ear infections. In twelve untreated normal control rabbits no evidence of *otitis media* was found. The author believes that these results indicate that *otitis media* is not blood borne, but takes place by direct extension from an upper respiratory tract infection.

Surface Tension of the Blood Serum in Syphilis.

ELIZABETH M. YAGLE (*Journal of Immunology*, January, 1929) has reviewed some of the recent work on the surface tension of the blood serum in health and disease and has undertaken a study of the surface tension of blood serum in syphilis because it is thought that the changes in syphilitic sera are of a physico-chemical nature and that complement fixation and precipitation reactions are due in part at least to changes in the surface tension of the sera. So far as possible the method of du Nouy was followed and the sera for study were chosen on the basis of their response to complement fixation and precipitation tests rather than on the basis of clinical diagnosis. The surface tension was measured within twenty seconds of the dilution of the sera and then again two hours later, the initial surface tension and the static surface tension respectively being thus determined. The time drop was determined by subtracting the static from the initial surface tension. The average time drops of twenty-five non-reacting and twenty-five reacting sera, heated and unheated, show that the time drop is slightly greater in reacting than in non-reacting sera and also that the time drop is slightly greater in heated than in unheated sera. Study of individual sera shows that there may be a greater difference in time drop between individual sera of the reacting or of the non-reacting group than between reacting and non-reacting sera. The drop of surface tension is not correlated with response or failure of response to complement fixation and precipitation tests and therefore surface tension determinations are of no value as a means of differentiating between syphilitic and normal serum. The so-called "syphilitic antibody" or reagin is evidently not highly surface active or at least not more highly active than other substances found in normal as well as syphilitic serum.

HYGIENE.

Post Mortem Bacteriology.

ROBERT DONALDSON (*The Journal of State Medicine*, September, 1928) states that the period which elapses between the time of death and the invasion of the blood and tissues by putrefactive organisms is highly important from the point of view of legal medicine. Under the conditions of English climate this period has a duration of thirty-six to forty-eight hours. During this interval it is possible to discover whether bacteria and what types of bacteria have played a pathogenic part during the latter part of the patient's life and within certain limits to ascertain whether such organisms are responsible for the person's death. There are other adjuncts to *post mortem* examination besides chemical analysis. Much may be learned from histological examination of the tissues, while a bacteriological investigation may throw an entirely new light on the cause of death. The author has collected from various sources the published results of cultural examination of the blood *post mortem* relating to 2,345 deaths. The blood was actually sterile in 1,177 of these, a percentage of 50·1, while organisms were grown in 1,168 or 49·6%. These organisms were for the most part pathogenic bacteria; only twice were putrefactive germs isolated. This offers fairly conclusive evidence that up to forty-eight hours at least the risk of contamination and overgrowth by putrefactive organisms may be regarded as negligible. Many have decried the value of *post mortem* bacteriological examination of the blood because they consider the organisms recovered merely represent *post mortem* invaders from the mucous surfaces of the respiratory tract or alimentary canal. If this be true, a diversity of microorganisms would be expected. The very reverse is actually found. In about 95% of the 1,168 bodies only one organism was found to be present and in nearly every instance that organism proved to belong to the pathogenic group. The opponents of *post mortem* blood culture argue that, since organisms may reach the heart blood *post mortem* from which they were absent during life, the presence of such in the blood after death cannot be an indication that septicæmia was present during life. To meet this objection and to avoid mistaking this local blood invasion for a general blood infection the collection of blood samples from the arm vein is advocated. To put this view to the test certain investigators made parallel cultures from the heart blood and arm vein blood. While bacteria were frequently obtained from the heart blood, they were only occasionally got from the blood of the arm vein. Moreover, when the peripheral blood contained bacteria, the heart blood contained the same organism and whenever bacteria were found in the peripheral blood the history indicated clearly that septicæmia had been present during life. By the simple device of collecting blood for examination from a peripheral vein, it may be concluded that if the blood be sterile, the patient did not die of septicæmia; conversely, if bacteria are recovered from the blood, there has been a general blood infection which has been the cause of death. This method of examination should be adopted as a routine measure in all sudden or unexpected deaths or when there has been any doubt about the clinical diagnosis.

Industrial Hygiene in Canada.

R. M. HUTTON (*The Journal of Industrial Hygiene*, October, 1928) states that following on the reports of the Health of Munition Workers Committee a federal committee on industrial fatigue was established in Canada in 1919. This committee was chiefly composed of the leading physiologists of the various Canadian universities. The committee functioned actively for about a year and a half, concentrating on the preliminary work of studying existing health conditions in Canadian industry, of

establishing liaison with industrial health agencies, both in Canada and elsewhere and of building up a reference library and information bureau on all aspects of industrial hygiene. It published a survey of general conditions of industrial hygiene in Toronto during 1920. The staff, equipment and library of the committee were then taken over by the Ontario Department of Health which instituted the Division of Industrial Hygiene for the promotion of industrial hygiene in Ontario. The division has been working for seven years and its activities embrace the carrying on and extension of the Committee's reference library and information bureau, together with propaganda and publicity work, the promotion of medical service in industry and the study and control of occupational diseases. In 1927 a somewhat cognate development, but from a different angle, has occurred in the Province of Quebec, a division of industrial hygiene having been formed in the Public Health Department of McGill University. The activities of this division are of three kinds, hospital, university and field. An industrial clinic is operated in the out-patient department of a large hospital and patients whose work is in certain dangerous trades, are studied there. Courses of lectures on industrial hygiene are being given to undergraduates, graduate nurses, social service workers and medical graduates. Medical service consisting of initial and periodical examination, treatment of accidents and minor illness, visiting nursing and plant inspection is being offered to industrial establishments. Thus the preliminary objectives of the Canadian Committee on Industrial Fatigue are at present being served from two major foci, a government department in Ontario and a university in Quebec. On the legislative side throughout the provinces general measures for the protection of health are incorporated in the *Factory Acts* and administered through factory inspection departments. There are no factory medical inspectors. Apart from such general control, legislation directly bearing on industrial health, such as exists in other countries, is very scanty in Canada.

Lacquer Solvents.

H. F. SMYTH AND H. F. SMYTH, junior (*The Journal of Industrial Hygiene*, October, 1928) summarize the results of animal tests made to determine the possible toxicity of solvents and diluents entering into the composition of nitro-cellulose lacquers, the concentration in which each may be safely used in a lacquer and the effects of lacquer solvents in general on the health. From their observations the authors would divide the solvents tested into three groups: Those usable with safety in concentrations usually employed or possibly in somewhat higher concentrations as ingredients of brushing or spraying lacquers. Ethyl acetate and amyl acetate and possibly butyl acetate would be placed in this class. Those usable with safety in present concentrations, but to be increased with caution as possibly harmful if increased materially. Here would be listed gasoline, turpentine and xylol. Those for which the present practice probably represents nearly the upper limit of safety in use as spraying lacquer constituents. In this group are toluol and butyl alcohol. Provided reasonable exhaust ventilation is assured, however, all these limits may be appreciably raised with impunity. The entire investigation furnishes a strong argument for the insistence on exhaust ventilation wherever lacquers of any type are being sprayed. The materials listed and tested do not exhaust the list of those used as lacquer materials and the fact that practice in this respect is still frequently changing and new ingredients continually being introduced, about which there is available no information as to their effect on the health, is another argument for the protection of the sprayer in all cases. The removal of benzol from lacquers will undoubtedly remove the greatest single hazard from either acute or chronic poisoning in lacquer work, but it does not warrant the discarding of precautionary measures.

Microbic Dissemination in Schools.

SHELDON F. DUDLEY (*The Lancet*, October 27, 1928) states that the spread of any species of living organism involves two processes. First, there is the necessity of finding transport for the dissemination of the organism in space and secondly, the species has to establish itself successfully in the new environment when found. A large group of parasites spreads in droplets of the secretions of the infected host. This method of bacterial dissemination is by far the most important in civilized communities because there are no practical methods of preventing pathogenic microbes from successfully using these droplets for their transport. The only way to avoid droplets entirely is complete isolation or the constant wearing of a respirator. Although the use of the latter is a wise precaution for those who have to attend patients with such virulent droplet infections as pneumonic plague or streptococcal influenza, yet it is scarcely feasible to keep a whole school or ship's company constantly in gas masks. The failure of isolation in checking the prevalence of infectious diseases is mainly due to two facts. First, the victims of many diseases are more infectious in the early than in the late stages of their illness and thus have usually passed their parasites on to others by the time they are segregated. The second reason is that many infectious diseases are accompanied by a multitude of hosts who show no evidence of infection. The importance of carriers lies in their elusiveness; they are often not suspected and even if they are, the laboratory examinations which may be undertaken for their discovery often do not yield positive results. Also the carrier retains his full mobility and mixes just as usual with his fellows. There is little definite evidence that vaccines have had much appreciable effect on the incidence of the minor respiratory diseases. It seems, therefore, that little impression can be made on the incidence of the minor droplet infections by attempts to isolate the distributors of them or increase the immunity of their receivers by means of a vaccine. Each individual of a herd has a special degree of resistance which can deal with quantities of bacteria below a certain minimum number. Any number of microbes less than the minimal infective dose will be destroyed by the host and is for him a subinfective dose. If more than the minimal dose is received instantaneously, infection is contracted at once. On the other hand, a subinfective dose is destroyed and no illness results. If, however, a further subinfective dose is received before the first is completely inactivated, successful infection will depend on whether the remains of the first dose, added to the second, exceed the minimal infective dose. The time necessary to contract infection is the minimal infective dose divided by the difference between the rate at which infective material is received and destroyed. This differential rate may be called the velocity of infection. Until the perfect vaccine materializes it is necessary to use any other method which may be available to combat the prevalence of droplet-borne infections. Although bacteria cannot be prevented from polluting the air, yet if the average distance between the members of the herd can be sufficiently increased, symptomatic infection will be considerably reduced and provided that the individuals in a community are not allowed to receive too many or too large droplets, good may actually result by an increased resistance of the herd as a whole to the spread of infection. The fundamental principle in preventing droplet infection in schools is linear space, aided by rapid change of air.

Action of Silica Dust in the Lungs.

P. HEFFERNAN AND A. T. GREEN (*The Journal of Industrial Hygiene*, October, 1928) state that up to the present time two different explanations have been offered, one physical and the other chemical, to account for the action of silica dust in the lungs. It soon became obvious that the physical theory (the hardness, sharpness and insolubility of silica dust wounding the lung substance) was inadequate. The second explanation, the chemical theory of Gye, Purdy and Kettle, is that insoluble silica becomes hydrolysed in the tissues and as a soluble protoplasmic poison is able to exert its action on the cells. If, however, silica is a soluble protoplasmic poison, how is it that there is "an enormously greater proliferation of the tubercle bacillus" in a silica coagulum in the tissues than in the same tissues in

the absence of silica? The authors adopt another interpretation, namely that the action of colloidal silica sol on animal tissues is essentially an adsorptive and coagulant one. That is to say, silica sol acts on animal tissues, not as a soluble protoplasmic poison, but simply as an active colloid, adsorbing fluids such as complement, serum and cell protoplasm; coagulating colloids of opposite sign, agglutinating or laking blood corpuscles according to the concentration or massiveness of the dose of colloid and producing all the phenomena described by Gye and Purdy, not because it is poisonous, but because it is a colloid of great surface energy.

Cancer and Tuberculosis.

RAYMOND PEARL (*The American Journal of Hygiene*, January, 1929) considers that the emphasis of medical research during the next generation at least is going to be in the direction of the general biological study of constitution in the broadest sense. The present day student of the problems of pathology has at his disposal the highly refined methods of physical and biological chemistry, of modern genetics, of cytology, of anthropometry and of modern statistical mathematics. The result is that the discussion of constitution in relation to disease is replacing the vague generalities of its prebacteriological stage with the precision of the basic sciences. The present study has to do with the constitutional differences between the tuberculous and the cancerous. The material comprised in 7,500 autopsies performed in The Johns Hopkins Hospital has been subjected to careful statistical analysis. There was first set up by a critically adequate procedure a case-for-case control series of persons who came to autopsy each one of whom was of the same sex, age, colour (race) as one of the 816 others who were found to have carcinoma, sarcoma or some other form of malignant tumour. Active tuberculous lesions were found at autopsy in only 6·6% of 816 bodies of persons having malignant growths and 16·3% of 816 bodies of persons without malignant tumours, but of the same race, sex and age as the former group. The active tuberculous lesions occurred over twice as frequently in the controls as they did in the malignant group in the present material taken as a whole. Active tuberculous lesions occurred 2·2 times more frequently among the controls of the carcinomatous than among those having carcinoma, whereas such active tuberculous lesions occurred 3·3 times more frequently among the controls of the sarcomatous than among those having sarcoma. Healed tuberculous lesions (apical scars, calcified tubercles in glands *et cetera*) occurred with equal frequency in the malignant disease and control groups, where they were taken as wholes or when carcinoma and sarcoma were separately treated. In each decade of age and in each sex and race division of the material, the percentage of persons with active tuberculous lesions at autopsy was definitely higher in the control group than in the malignant disease group. Looking at the matter from the reverse aspect, it was found that in this autopsy experience persons who had tuberculous lesions of any degree of activity whatever, showed a relative incidence of cancer and other malignant tumours only a little more than half as great as that exhibited either by persons with no tuberculosis discoverable at autopsy or those who had only old, healed tuberculous lesions. In 886 persons of both sexes and races compared who had a great deal of florid, active tuberculosis (acute, ulcerative tuberculosis of the lungs or intestines, tuberculous meningitis, caries of the vertebræ *et cetera*) there were but eleven malignant tumours or 1·2% of the total number. On the other hand, in 886 persons with no recorded lesions of tuberculosis at autopsy, having the same age, sex and racial distribution case for case as the very actively tuberculous, there were 82 malignant tumours or 9·3%. Among the females, both white and coloured, there was not a single malignant growth in those with much active tuberculosis. This general result was confirmed in each separate age, sex and colour group, save for a few instances in which the numbers were too small to give any reliable indications whatever. There was no significant difference in this material between groups of persons with cancer or other malignant tumours and groups of persons without any malignant neoplasms in respect of the incidence of either pleural adhesions or emphysema, all of the persons in both groups having no lesions of tuberculosis. The author concludes that there is a definite and pronounced incompatibility or antagonism between the two diseases, cancer and tuberculosis, of such sort that both occur together at the same time in florid activity in the same individual only with great rarity.

Spray Painters in Automobile Refinishing.

JEROME MEYERS (*The Journal of Industrial Hygiene*, November, 1928) examined a group of workers in the automobile refinishing trade during part of July and August, 1927. In all twenty-four men distributed in five shops were subjected to complete physical blood and urinary examinations. In the automobile refinishing trade, which usually includes not only sprayers, but also painters, upholsterers, metal workers, woodworkers, assemblers, rubbers and helpers, there is constant exposure, either direct or remote, by inhalation or contact, to such chemicals as turpentine, gasoline, naphtha, lead, methyl alcohol, amyl acetate, benzol, toluol and other volatile agents. In this study particular attention was directed to benzol. Distinct physical defects attributable to benzol were very evident from this study. Of the twenty-four men examined seven or 29·1% had a blood picture with an abnormally low leucocyte count. This finding was not confined to the men directly exposed to the spray. An abnormally high leucocyte count, high red blood cell count and decreased hæmoglobin percentage were also in evidence. In five workers or 20·8% the spleen was palpable—a surprising percentage and a condition not before clinically reported in such an investigation. Pallor was common, though in some instances with high red counts the complexion was ruddy. Chronic bronchitis, phthisical appearance and naso-pharyngeal irritations were also in evidence. Physical appearance, length of exposure and the presence or absence of subjective complaints are not indicative of the presence or absence of blood or physical changes or of their severity. Sprayers immediately exposed to sprays or those indirectly exposed may be affected. The blood picture and physical condition before this trade is entered must always be kept in mind.

The Training of a Public Health Nurse.

EDITH K. RUSSELL (*The World's Health*, October, 1928) considers the terms "public health nurse" and "public health nursing" as clumsy and confusing. No less than five distinct occupations are embraced, namely, bedside nursing, school nursing, midwifery, social work and health visiting. Training for public health nursing should include a thoroughly sound preparation for bedside nursing, but such training need not and should not be given as a preparation for hospital nursing. The second and equally important aspect of a pupil's preparation is the study of health, its conditions and requirements. This study may be grouped under three headings, namely, physiological, fundamental to a scientific understanding of public health work; psychological, mental hygiene being essentially and inextricably a condition of health; preventive, the specific contributions of bacteriology and immunology having made possible some of the chief triumphs of public health work. The present one-year course of trying to teach public health work in a few months to a group of students who have received no scientific foundation for an understanding of that work, is unsound and the student finds at the end of that time that she has no adequate foundation upon which to build. The new four-year course started last year in Toronto is an effort to decide upon the full training needed by a public health nurse. The first year of this course is spent at the university in the study of certain foundation work of science. The second and third years are spent in the School of Nursing of the Toronto General Hospital following the required training (with certain special arrangements) for the school's diploma in hospital nursing. The fourth year is spent at the university in a study of organized public health nursing.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Brisbane Hospital on February 1, 1929, DR. MERVYN PATTERSON, the President, in the chair. The meeting took the form of a series of clinical demonstrations by the members of the honorary staff.

Coronary Thrombosis.

DR. W. N. MARKWELL reported a case of coronary thrombosis. The special interest in this instance was that at the time of the attack ten months previously the patient was only thirty-eight years of age and there had been no premonitory symptoms nor had syphilis been detected. The retinal arteries were reported to be normal, but the condition was held to be one of arteriosclerosis. The patient had undergone considerable mental stress since the war. At the present time he was comparatively well, helping in the ward, but occasionally suffered from shortness of breath, even with such a sheltered life. The prognosis was bad. Questions were asked and answered concerning the symptoms, prognosis and treatment of the condition.

DR. S. F. MCDONALD said that patients suffering from coronary thrombosis, on account of collapse, pain in the epigastrium and rigid abdomen frequently fell into the hands of the surgeon, with sad results.

Urological Conditions.

DR. CHARLES M. LILLEY gave a demonstration on three urological conditions: (i) Renal tuberculosis in a kidney with a double ureter; (ii) collection of large stones (12) at the lower end of the ureter; (iii) a ureteral kink in a patient previously operated on for appendicitis.

Tumour of the Testicle.

DR. NEVILLE SUTTON showed a patient, aged sixty-eight years, an engine driver, married, who had been born in Glasgow. He had been admitted to hospital on December 24, 1928, complaining of swellings in the groins and swollen testicles. No history of venereal infection had been obtained and there had been no previous illness of note. The patient had been torn with a wool hook in the right groin and had a small scar. He had one child, but his wife had had four or five miscarriages.

The patient had had a swelling in the right groin for the past eight or nine months. At first it had appeared when he was lying down, but for the last few months it had been present continuously. It was not painful or tender. Three weeks prior to admission both testicles had started to swell but they had not been painful. They had been treated by hot fomentations and had become smaller. He had had no urinary symptoms and the action of his bowels was regular.

On examination he was seen to be rather a thin man in moderately good general condition. There was a hard swelling in the right inguinal region, not tender, not reducible into the abdomen, presenting no impulse on coughing and in the line of the spermatic cord in the inguinal canal.

The right side of the scrotum was swollen and felt cystic and was translucent. The left side of the scrotum was larger and harder and more nodular with a fusiform swelling extending up the cord into the inguinal canal. There was no tenderness on palpation. *Per rectum* the prostate felt normal, but there were one or two small seed-like nodules felt on the left side.

The left pupil was slightly larger than the right and both reacted to light. His knee jerks were sluggish, but equal. The superficial abdominal reflexes were present. His blood had not reacted to the Wassermann test. On December 28, 1928, operation had been performed on the right side through an inguinal incision and a firm solid tumour had been found, about 7·5 centimetres (three inches) in diameter, incorporated in the spermatic cord fairly high up in the inguinal canal. There was a small amount of clear fluid in the right *tunica vaginalis* and the testis looked normal. The cord had been ligated at the abdominal ring and cut and the cord, tumour and testis removed. This specimen was exhibited.

A section of the tumour of the cord had been examined and reported on as a so-called alveolar sarcoma, composed of large round cells of epithelioid type, probably an adult embryoma. A section of the testis appeared normal. Subsequent to the operation the patient had developed a transient right facial palsy, but this had cleared up. It had apparently been a Bell's palsy (peripheral neuritis). The wound had healed without incident. Two days previously the left scrotum had been aspirated and about fifteen centimetres (half an ounce) of deep yellow fluid obtained. The nodular tumour of the left testis could be felt.

Dr. Sutton said that tumours of the testicle were rare; they were estimated to be found in about one in one thousand five hundred of male patients admitted to surgical beds in London hospitals. No subject in the entire range of surgical pathology had provided more diversity of opinion than the interpretation and attempted classification of new growths of the testicle.

F. Gordon Bell in *The British Journal of Surgery* of 1925 had stated that the vast majority of tumours fell into two main groups: (i) the teratoid group, (ii) the germinal celled variety *teratoma testis* might be defined as a tumour composed of elements derived from all three layers of the blastodermic vesicle, in differing degrees and varying grades of complexity. In many cases, however, representatives of only two layers were present and probably frequently only one layer was represented. One school of pathologists tended to extend the scope of *teratoma testis* and so regarded all testicular tumours as of teratoid origin. Ewing was amongst their number.

Shaus considered there was sufficient evidence to support the theory that a definite group of tumours originated from the seminiferous cells of the tubules and considered that seminoma or mesothelioma was the best name for it. Its distinguishing features were (i) that it most often appeared in the fourth decade of life; (ii) it grew more slowly than the teratoid variety; (iii) carcinomatous features usually prevailed, but mixed histological characters might be found in the same tumour; (iv) it presented a specific structure definitely related to the seminiferous cells; (v) metastases occurred by way of the lymphatics, but occasionally by the blood stream. A left-sided orchidectomy was to be done in this patient, but the prognosis was not very good.

Swelling of the Testis with Nervous Signs.

DR. ALAN E. LEE showed a patient who was suffering from bilateral testicular swelling associated with nervous signs. This patient, a previously healthy young man, aged nineteen years, had been admitted to hospital complaining of certain nervous signs, especially weakness of certain muscles of the left shoulder and proptosis of the right eye. In the course of a routine examination it had been discovered that a scrotal swelling was present. Dr. Lee's advice had been asked as to the nature of this swelling and its relation, if any, to the nervous signs.

Questioning had disclosed the following history. Eight weeks previously rapid swelling had occurred in the right side of the scrotum, followed within a week by swelling of lesser extent on the left side. As an apparently similar swelling had occurred two years before on the right side and had completely subsided in six or seven weeks, the patient had not attached great importance to the matter. But a week or so later the muscles of the left shoulder had become sore and painful and after several days of this soreness weakness had developed so that he was unable to abduct the arm from the side. About a week later, four weeks before the date of the meeting, after a few days' headache definite proptosis had rapidly developed in the right eye, together with double vision. This had continued up till the present time and moreover obvious wasting had occurred in the affected deltoid muscle.

On examination of the scrotal swelling, it had resolved itself into a typical tense fluctuant swelling within the *tunica vaginalis*, on each side, clinically resembling hydro-

cele except for the absence of translucency. Exploration with a large bore canula on either side had failed to evacuate any fluid, only a little blood being obtained. The provisional diagnosis of filarial hydrocele therefore could not be sustained and one of bilateral hæmatocele was suggested.

To clear up the question, however, it had been decided to explore the swelling. Only a small amount of straw coloured fluid had been present in the tunica, the mass of the swelling on the right side being very enlarged testis and epididymis. A section of the testis had been removed for microscopical examination and the scrotal incision closed. Two days later, however, owing to persistent oozing, the right testis had been removed. The pathological report on the testis was "small round celled sarcoma."

While Dr. Lee did not altogether disbelieve the diagnosis of sarcoma, from the clinical point of view he offered the following objections: The onset had been sudden, the tumour reaching its maximum size in a few days and since then it had if anything retrogressed. The association of swelling of the epididymis, which microscopically resembled that seen in epididymo-orchitis, was very difficult of explanation if this diagnosis were to be accepted. But acceptance of this diagnosis would also call for a similar explanation for the opposite testis and this would presuppose a lymphatic spread from one testis to the other, *viâ* the aortic lymph glands within two weeks of the onset of the tumour and a blood spread, apparently missing the lungs within four weeks of the primary origin, the nervous signs being regarded as due to multiple sarcomata.

For all these reasons he inclined to the diagnosis of a septic epididymo-orchitis of non-urethral origin for the scrotal tumour and was unable to explain the nervous lesion. In favour of this diagnosis was a white cell count in the blood of 35,000 per cubic millimetre. Of the leucocytes 84% were lymphocytes, 15% neutrophile cells and 1% large mononuclear cells.

DR. A. P. MURPHY asked whether the spread of the growth was considered to be a vascular one and whether an X ray examination of the chest had been made.

DR. J. V. DUHIG considered that the boy was suffering from a sarcoma and that there was no evidence of inflammatory trouble. Any irritation would cause liquefaction and the naked eye appearance did not to his mind resemble inflammation.

Under the microscope the specimen showed a single type of cell which was not derived from the blood or from the formed tissue cells of the organ involved. There were also a few eosinophile reticular cells and he considered it to be a lymphosarcoma. The structure of the tumours in the testis and epididymis was exactly similar.

Dr. Duhig remarked that these tumours were very rare, affected young persons and were bilateral, rapid in onset and growth. Ewing had seen only two examples. The patient probably ran a temperature in the early stages. He questioned whether this was the primary growth or whether it was secondary to a lymphosarcoma of the abdominal lymphatic glands.

DR. J. BOSTOCK said that he had seen this patient privately and had sent him into hospital for further investigation. It was noteworthy that he had made no complaint whatever about the testes and involvement of these had been discovered during routine examination. He stated that a one-sided exophthalmos was generally due to either chloroma, sarcoma or cavernous sinus thrombosis. It was difficult to account for the weakness of the deltoid muscles in view to the fact that the skiagram revealed no secondary deposits. He considered that filaria might have been a possible cause, but examination of the blood after midnight showed that no filaria were present. In view of the pathological findings and blood picture they had to consider that the muscular wastings and eye signs were due to hæmorrhage occurring in a lymphosarcoma.

DR. NEVILLE SUTTON said that on questioning the patient information had been elicited which suggested that probably the right testicle did not return to normal after the first swelling two years previously.

DR. S. F. MCDONALD called attention to the enlarged lymphatic glands in the groin and axilla, the enlarged spleen and the petechial spots on the abdomen. He would suggest that swelling of the tissues round the eye was due to hæmorrhage into them and that a swelling into the tissues of the neck had caused pressure on the cervical plexus, with resulting weakness of the muscles. He would question whether these lymphosarcomata were not closely allied to the lymphocythæmias in which enormous local deposits of lymphatic cells often occurred.

DR. J. LOCKHART GIBSON thought that the proptosis of the eye was due to hæmorrhage into the tissues behind it.

DR. ELLIS MURPHY agreed with Dr. McDonald. There were abnormal types of lymphatic leucocythæmia which were not yet classified. He would suggest the making of another blood count with particular attention to the white cells.

DR. L. M. MCKILLOP supported Dr. Duhig's diagnosis of lymphosarcoma, the reasons in its favour being (i) the age of the patient, (ii) the bilateral nature of the swelling, (iii) the absence of any known cause of swelling of the testis such as sepsis *et cetera*, (iv) the microscopical appearances which were typical of a lymphosarcoma.

He considered that leuchæmias were really forms of sarcomata of the blood and that this was a local manifestation.

Dr. Lee, after hearing the discussion, was inclined to agree with the view that the patient was suffering from an acute leuchæmia. Further examination of the blood would be carried out and the chest would be examined by X rays for metastases or glandular masses. The serious state of this patient after two short anæsthesias certainly suggested the presence of the lymphatic diathesis, if such a condition existed.

In spite of the weighty evidence adduced by Dr. Duhig and others in favour of lymphosarcoma, he felt that the clinical cause of the diseases and especially the undeniable fact that the lesions were retrogressing, almost precluded the diagnosis.

Decortication of the Lung for Chronic Empyema.

Dr. Lee also showed an adult male patient who had been operated on for empyema at another hospital ten months previously, a rib resection with drainage having been performed. As a persistent sinus had remained, the patient had been transferred to the Diamentina Hospital for Chronic Diseases, from which he had been admitted to the Brisbane General Hospital for further treatment.

A large cavity had been present in the posterior part of the pleural cavity, extending from the diaphragm almost to the apex of the chest. At a first operation the ribs had been extensively removed around the region of the sinus till a view of the complete cavity was obtained. It had appeared that the chief bar to further expansion of the lung was the light, drum-like visceral pleura stretched from the posterior to the lateral wall of the chest cavity. A week later the posterior limit of the original V-shaped incision had been extended upwards to the level of the spine of the scapula and about 7·5 centimetres (three inches) of each rib up to the third had been removed, together with the intercostal structures and parietal pleura which was at least 1·25 centimetres (half an inch) thick. The visceral pleura had then been stripped completely from the lung over the area of the empyema cavity, this resulting in an immediate expansion of the lung almost sufficient to obliterate the space. One flap had been sutured into place with interrupted silk worm gut sutures. While the greater part of the incision had healed well, the lower part had broken down badly.

The expansion of the lung had been such that Dr. Lee was hopeful it would result in a cure of the condition. In the event of only partial success, an anterior osteotomy of the divided ribs would allow partial collapse of the chest wall, converting the operation into a modified Estlander's operation.

Gastric Ulcer.

Dr. Lee showed a portion of a stomach removed three days before for gastric ulcer.

The patient, aged thirty-seven years, had had a perforated ulcer three months before; he had been picked up in

the street by the ambulance in a state of collapse and had been operated on by Dr. Lee within one and a half hours of the perforation. After he left hospital, his dyspeptic symptoms had rapidly recurred and he had been readmitted for further treatment. At the second operation a large area of thickening had been present high up in the lesser curvature. There had been dense adhesions to the pancreas and the amount of stomach finally removed had included practically the whole of the lesser curvature and altogether at least three-quarters of the stomach.

The base of the crater was shown to be formed almost entirely of extragastric structures and it seemed that no method of treatment which did not include removal of the ulcer would have produced a cure.

The patient's condition at the time of meeting was quite satisfactory.

Nephritis.

DR. A. J. REYE showed two brothers suffering from nephritis.

The first, a boy of thirteen and a half years, had been admitted on October 8, 1928. His past history was that he had been treated for plumbism and kidney trouble at the Children's Hospital when aged about seven and for kidney trouble previously.

On admission his facial appearance had been typical of chronic nephritis. The systolic pressure had been 145 millimetres of mercury. Albumin had been present in his urine. On October 11, 1928, the blood urea had been 121 milligrammes and the figures obtained by the urea concentration test 1·3, 1·1 and 1·2.

The erythrocytes had numbered 4,000,000 per cubic millimetre and the hæmoglobin value had been 75%. No punctate basophilia had been found. Medical diathermy (auto-condensation) had been commenced on December 4, 1928.

On December 20, 1928, the blood urea had been 35 milligrammes and the figures obtained by the urea concentration test 0·8, 1·8 and 2·1. He had been discharged to the Out-patients' Department for medical diathermy on December 23, 1928. On January 29, 1929, the blood urea had been 29 milligrammes and the figures obtained by the urea concentration test 0·7, 0·9 and 2·0.

The brother of the last mentioned patient was fifteen years old. He had been admitted on October 20, 1928, suffering from œdema of face, legs and abdomen. His systolic blood pressure had been 160 milligrammes of mercury. The urine had had a specific gravity of 1022 and it had contained albumin and a trace of blood. On October 26, 1928, the blood urea had been 54 milligrammes and the figures obtained by the urea concentration test 0·9, 1·0 and 1·2.

Medical diathermy (auto-condensation) had been commenced on December 5, 1928. On December 20, 1928, the blood urea had been 16 milligrammes and the figures obtained by the urea concentration test 1·1, 2·0 and 2·9. On December 23, 1928, the patient had been discharged to the Out-patients' Department for medical diathermy. On January 29, 1929, the blood urea had been 23 milligrammes and the figures obtained by the urea concentration test 1·5, 2·3 and 2·0.

Dr. Reye said that a sufficiently long period had not elapsed to justify any opinion as to the effect of diathermy, but they were continuing treatment. The aim of the treatment was increased urea elimination and relaxation of the arterioles and thus the breaking of the vicious circle.

DR. C. A. THELANDER, commenting on the cases of nephritis reported by Dr. Reye, inquired whether the statement in the history of one of the brothers, that he had had lead poisoning, had been given any consideration. In his experience if one member of a family had plumbism, there was always a likelihood that a brother or sister of "adjacent" age was also affected. It seemed quite possible that both of these patients were suffering from nephritis of plumbic origin.

If that were so, whatever benefit might be secured by other means, it was surely absolutely essential to see whether lead was still in the system and if it were present to attend to its elimination.

He had used the process known as deionization very extensively principally as a test for the presence of lead with the cooperation of Mr. Henderson, the Government analyst, in some cases and of Mr. Chater in others. He had no doubt as to the efficiency of the test. The urine test for the presence of lead was unreliable, unless preceded by the administration of iodide of potash, which was rather dangerous in some conditions.

DR. L. J. NYE considered that diathermy was very useful in cases of diffuse glomerulo-nephritis, but doubted if its results were more than temporary in cases of so-called chronic interstitial variety in which there was definite sclerosis of the vascular endothelium. Nevertheless as they had no method of treating such patients with advantage, he suggested that all those of this type at the hospital should be passed over to Dr. Reye, so that he could demonstrate the results of treatment in a large series.

DR. J. LOCKHART GIBSON said that when Oliver had introduced deionization, he was particularly pleased because if Oliver's findings were confirmed, the chronic nephritis in the later lives of the children who had suffered from lead poisoning and attributed by many of his colleagues to it, could be prevented. The few cases in which he had made observations appeared to confirm Oliver's findings, but he had retired from the Hospital for Sick Children before he was satisfied. He wished that further observations might be made. He did not think the matter settled either for or against deionization. It was possible that the conflicting evidence might be explained by the state of the lead in the body. If it were in a colloid state, deionization would not be expected to remove it. If it were in a crystalloid state it might be expected to do so. This also might explain the fact that in patients suffering from lead poisoning the urine might at one time contain no lead while at other times lead was found in it.

DR. A. P. MURPHY said that the latest writings showed that the world's authorities considered the rate of tissue penetration of heavy metallic ions so slow that it was a physical and chemical impossibility to extract lead by deionization.

DR. S. F. MCDONALD said that these children had been seen fairly early. He found that children with a urea concentration of 1·0 with care, treatment and a happy, easy life, lived on very well, often for some years. If they were overfed or overclothed they began to go downhill straight away. One point to which he would like to draw attention in the light of researches detailed in THE MEDICAL JOURNAL OF AUSTRALIA, was the high lead content of eggs. It had always been a set rule that eggs should not be given to nephritic patients and this might not be the mere medical superstition it had always appeared to be, but might actually have some foundation in fact.

Fracture of the Spine.

DR. S. A. C. DOUGLAS showed a patient who was suffering from fracture of the spine. She was a woman, aged twenty-seven years, who had been thrown from a car and had fallen on to her left shoulder. She had had a compression fracture of the eleventh thoracic vertebra and no interference with the cord functions.

This patient was shown to illustrate the mechanism of compression fracture and the site at which fracture usually took place, following indirect violence. On examination there had been a severe contusion over the left scapula and a tender acutely angled prominence at the level of the eleventh and twelfth thoracic vertebræ. The maximum tenderness had been over the interspinous ligament between the eleventh and twelfth thoracic vertebræ. This patient also illustrated the method of reduction of fractures of this type. The bed was rendered unyielding with fracture boards, the patient was laid flat with folded towels on each side of the spine, the upper edge of the towels level with the greatest prominence of the spine and just sufficiently far apart to clear the spinous processes. On the next morning the towels were raised and the legs were extended by taking away all support from under the knees. The head was lowered as far as possible without alteration of the curve of the neck.

By the third day the deformity was quite reduced, there was no pain and at the end of a week the patient was

transferred to a Bradford frame, bent in at site of fracture. She would be in this frame till consolidation took place, when she would be allowed up on a Taylor's brace.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Society's Rooms, Tasmanian Museum, Hobart, on February 23, 1929, DR. D. H. E. LINES, the President, in the chair.

Annual Report of Council.

The Honorary Secretary, DR. J. H. B. WALCH, presented the annual report of the Council.

The Branch Council has pleasure in submitting the following report of the work of the Branch for the year ended February 23, 1929.

Membership.

From seventy-three at the end of last year the membership has increased to eighty-three. Three members have been transferred to other Branches, while six have moved into our area. Seven who had been crossed off as unfinancial, have been reelected after paying the amounts owed and one new member has been elected.

Meetings.

Nine ordinary general meetings were held during the year besides the annual meeting and one special general meeting. The average attendance was 12·4.

Papers.

The following papers were read during the year:

February 18.— Dr. E. H. Hogg: Presidential Address.
May 8.—Dr. F. W. Fay: "Hospital Management."
June 12.—Dr. H. W. Sweetnam: "Some Recent Work on the Physiology of the Liver."
July 17.—Dr. H. W. Sweetnam: "The Removal of Pelvic Urethral Calculi by the Intraabdominal Route."
October 9.—Dr. E. Brettingham Moore: "Some Notes on Analgesic Labour."
November 13.—Dr. A. W. Shugg: "Ulcers of the Leg."

A number of cases were demonstrated and discussed and several meetings were occupied with discussion on the Hobart Public Hospital negotiations.

Meetings of the Branch Council and Attendance.

Eighteen meetings of the Branch Council were held, the average attendance being 5·6.

The attendance of members of the Branch Council was as follows:

The President, Dr. D. H. E. Lines	13
President-elect, Dr. G. E. Clemons	0
Vice-President, Dr. Newell	0
Honorary Treasurer, Dr. E. A. Rogers	11
Honorary Secretary, Dr. J. H. B. Walch	18
Elected members—	
Dr. W. Giblin	7
Dr. T. Butler	12
Dr. F. W. Fay	14
Dr. A. W. Shugg	12
Dr. S. Gibson	15

Work of the Branch Council.

Much time was spent by the Branch Council in discussion of the Hobart Public Hospital problem and attempts to find a solution which might be acceptable to the Branch and the Hospital Board. In this connexion our thanks are due to Dr. Fay for his vigorous and tactful handling of the situation as the representative on the Hospital Board of the Hobart medical practitioners.

In response to an inquiry from New South Wales the Branch Council obtained a report on the working of the *Mental Defectives Act* in Tasmania.

The Branch Council reconsidered the information, obtained by a previous council, on the ethical position of homœopathy at the present day and after consultation with the Northern Division decided that there is no ethical objection to members attending patients at the Launceston Homœopathic Hospital.

The advisability of incorporating the Branch as a company with limited liability was considered, and opinions obtained from the Branch's solicitors and from Dr. R. H. Todd. The Branch Council decided to make no recommendation in the matter for the present.

The Branch Council has discussed in detail the draft revised rules which they now unanimously recommend for adoption by the Branch.

Subcommittees.

Fees Subcommittee.—Personnel: Dr. T. Butler, Dr. F. W. Fay, Dr. A. W. Shugg and Dr. S. Gibson.

This committee was formed to revise the scale of fees recommended for the use of members. The revised scale which differs very little from the old one, was adopted by the Branch Council, with the intention of printing it with the revised rules.

Hospital Subcommittee.—Personnel: Dr. W. Giblin, Dr. H. Butler, Dr. F. W. Fay and Dr. J. H. B. Walch.

These members were appointed to meet delegates from the Hobart Public Hospital Board. Various proposals for staffing the hospital were discussed at the meetings without any satisfactory conclusion being reached, when further discussion was postponed until publication of the report asked for by the Government from Mr. R. J. Love.

Post-Graduate Subcommittee.—Personnel: Dr. W. E. L. H. Crowther, Dr. A. W. Shugg and Dr. E. A. Rogers.

This committee was formed with the object of arranging lectures by visiting doctors. It is to their activity that we owe the arrangements for the course of lectures at present in progress.

Representation on Federal Committee.

For the year ending December 31, 1928, the Branch was represented on the Federal Committee of the British Medical Association in Australia by Dr. Gregory Sprott and Dr. E. Brettingham Moore.

The representatives elected for 1929 are Dr. Gregory Sprott and Dr. A. W. Shugg, these being the only members nominated.

The report was received and adopted.

Election of Office-Bearers.

The President announced the result of the election of office-bearers and members of the Council for the ensuing year as follows:

President: Dr. G. E. Clemons.
President-elect: Dr. T. Butler.
Honorary Treasurer: Dr. J. S. Reid.
Honorary Secretary: Dr. J. H. B. Walch.
Members of Council: Dr. F. W. Fay, Dr. S. Gibson, Dr. A. W. Shugg, Dr. G. Sprott, Dr. H. W. Sweetnam.

It was noted that no member had been nominated for the position of Vice-President. The President declared the position vacant.[1]

Branch Rules.

A new set of rules for the Branch had been drafted by the Council and was submitted to the meeting. It was pointed out that the Council had given the draft rules detailed consideration. The new rules were adopted unanimously and it was resolved that the existing rules should be annulled as soon as the approval of the Council of the British Medical Association to the new rules had been received.

Induction of President.

DR. LINES introduced DR. G. E. CLEMONS as the President.

THE ANNUAL MEETING OF THE WESTERN AUSTRALIAN BRANCH was held at the Hospital for the Insane, Claremont, on March 24, 1929, DR. J. BENTLEY, M.C., the President, in the chair.

[1] At a subsequent meeting of the Council Dr. F. W. Fay was appointed Vice-President. Dr. A. Giblin was appointed a member of the Council in place of Dr. Fay.

Financial Statements.

The Honorary Treasurer, DR. D. D. PATON, presented the balance sheet and financial report for the twelve months. These were received and adopted.

President's Address and the Annual Report.

In accordance with custom, the retiring President combined his address with the report of the Council.

Membership of the Branch.

The membership of the Branch has increased from 228 to 234. It is with very deep regret that the President has to record the death of two members of the Branch, namely Dr. B. F. Hussey, of Toodyay, and Dr. L. F. West, of Northcliffe.

Meetings.

The meetings of the Branch have been well maintained. There were eight general meetings with an average attendance of thirty-one members. These meetings included clinical evenings kindly arranged by Dr. C. W. Anderson, of the Perth Hospital, and Dr. L. G. Male, at the Children's Hospital. Interesting papers were read by Dr. A. W. Farmer, Dr. R. C. E. Atkinson, Dr. F. Clark and Dr. S. E. Craig.

Meetings of Council.

The Council held eight meetings with an average attendance of six members. Much work was done both by the general meetings and the Council. The following are the main items considered.

Annual Subscription.

The annual subscription of members has been increased by five shillings for the purpose of reducing an overdraft at the bank in the name of the Australasian Medical Publishing Company, Limited, guaranteed by some of the Directors individually and collectively.

Junior Resident Medical Officers.

Inquiries were made into the salaries paid to junior resident medical officers of hospitals in Australia. As the salaries paid in this State were as good or better than those paid in the eastern States, it was decided to let the matter stand.

Nomination for Membership of the Council.

The nomination paper for membership of the Council has been altered this year; it provides that the signature of both the proposer and the seconder must be appended, together with a statement that the member nominated for office has consented to stand, if elected.

The Workers' Compensation Act.

The Workers' Compensation Act was considered at many meetings; a fresh schedule was considered and approved; this was also endorsed by the Underwriters' Association. It is said to be giving satisfaction. In dealing with this subject the President wishes to state that the Medical Special Committee had given splendid service in connexion with disputed compensation accounts. Although much comment has been made of the great benefit of *The Workers' Compensation Act* to the medical profession, which it undoubtedly is, and also the adverse comment that members of the profession were making a welter of the opportunity of charging, only 112 cases have been submitted to the Special Medical Committee for revision to date. The thanks of the profession are due to this subcommittee which has saved the members of the Branch a considerable sum of money, as undoubtedly a certain number of the cases which have been adjusted, would have ended in costly litigation. The cooperation of every member of the profession is earnestly sought, so that the subcommittee may continue its good work.

The Anatomy Act.

The Minister of Public Health has agreed to submit an anatomy act to Parliament. The Council's representatives, supported by the University and the Dental Association, approached the Minister. They were ably assisted by the Principal Medical Officer, Dr. Atkinson. There was not time to get the bill presented to Parliament last session, but the Council hopes that an anatomy bill will be presented this session.

Friendly Society Practice.

It was hoped that a model lodge agreement for the whole of Australia, as approved of by general meeting, would be in force, but as this model agreement for Australia has not yet been satisfactorily completed in the eastern States, it is not proposed to make any alteration in this State, but to carry on with the present model lodge agreement which is working satisfactorily.

Australasian Medical Congress (British Medical Association).

Members are reminded that the third session will be held in Sydney in 1929; those who are desirous of attending, are requested to communicate with Dr. J. J. Holland, the local Honorary Secretary.

Branch Building.

It was suggested that the Branch should own its own building. A special committee was formed by the Council; inquiries were made into land values and property offering. Nothing suitable to date within the Council's means has been offered.

Visit of Dr. Earle Page.

A special meeting of the Council met Dr. Earle Page when he last visited Western Australia. This visit was in an unofficial manner. At the same time there was a discussion on the national insurance bill which, it is hoped, will be ultimately beneficial to the profession.

Annual Dinner.

The Council decided that to hold an annual meeting in December was holding it at the wrong time of the year; that the weather was generally hot and it was agreed that one of the winter months would be best for this purpose. It was decided that the next annual dinner will be held during the centenary celebrations.

Hospital Bill.

This very important matter is receiving much consideration and comment at present. A hospital bill to find funds to run public hospitals and also subsidized private hospitals for intermediate wards failed to go through Parliament last year, but will most likely be brought up again next session.

It appears to the President that without doubt it is the duty of the Government to maintain the public hospitals for the treatment of sick poor and the fact is invariably lost sight of that the medical officers of the staffs in these institutions give their services in an honorary capacity. It certainly appears that intermediate hospitals are absolutely necessary for that class of patient who is not rich enough to pay full fees for medical attendance and private hospital, while on the other hand they are not destitute and deserving of free medical attendance from the medical staff. The suggestion to have intermediate wards in public hospitals is undesirable, even as a temporary measure, as immediate difficulties will be created by some patients being treated by the honorary staff free and other patients being entitled to their medical attendant, whoever he may be, following them into the intermediate ward.

The primary object of our public hospitals for which the honorary staffs give their services for nothing, is that only destitute cases are admitted and our public hospitals are not convalescent homes nor should patients occupy beds who are in a chronic condition. If this principle was adhered to the expenses of running the public hospitals would be greatly reduced. It is admitted that motor accident cases have greatly increased the number of urgency beds necessary, but these cases should be removed from public hospitals where they are able to pay, immediately it is safe to do so.

Election of Office-Bearers.

The President declared the result of the election of the office-bearers and members of the Council for the ensuing year as follows:

President: Dr. J. J. Holland.
Vice-President: Dr. H. B. Gill.
Ex-President: Dr. J. Bentley, M.C.
Honorary Secretary: Dr. R. H. Crisp.
Honorary Treasurer: Dr. A. Syme Johnson.
Members of Council: Dr. T. L. Anderson, Dr. D. P. Clement, Dr. M. K. Moss.

It was stated that under the revised ethical rules the Ethical Committee, being a subcommittee of the Council, would be appointed by the Council.

Induction of President.

DR. J. BENTLEY then introduced DR. J. J. HOLLAND as the President of the Branch.

Election of Auditors.

The President announced that Dr. E. C. Dean had intimated that he would be unable to continue his services as honorary auditor to the Branch. It was resolved to tender the thanks of the Branch to Dr. Dean for the services he had rendered in the past.

On the motion of DR. J. BENTLEY, seconded by DR. D. P. CLEMENT, Dr. A. E. Randell and Dr. S. E. Craig were unanimously elected honorary auditors for the ensuing year.

Librarian.

DR. F. J. CLARK read the report of the librarians. Dr. Clark and Dr. G. R. Troup stated that they did not desire to be reelected as librarians. On the motion of DR. H. B. GILL, seconded by DR. S. E. CRAIG, Dr. R. D. McKellar Hall and Dr. J. G. Hislop were elected librarians.

Representation of the Branch.

DR. G. R. TROUP and DR. F. J. CLARK were appointed delegates of the Branch at the annual meeting of the British Medical Association to be held in July in Manchester. It was resolved to leave the appointment of a representative to attend the Representative Meeting to the Council.

Benevolent Fund.

On the motion of DR. H. J. GRAY, seconded by DR. J. E. FERGUSSON STEWART, it was resolved that the Council be asked to take steps to institute a benevolent fund as a private body.

The Medical Journal of Australia.

DR. J. G. HISLOP moved that all papers read at meetings of the Branch, being the property of the Branch, should be published in THE MEDICAL JOURNAL OF AUSTRALIA. He pointed out that the journal according to the constitution of the Australasian Medical Publishing Company, Limited, was the official organ of the Branches of the British Medical Association in Australia. He wished the principle to be established that the property of papers read at meetings of the Branch was vested in the Branch and that the journal should be used as the medium for the publication of the transactions of the Branch. It was resolved to refer the matter to the Council.

Condolence.

The President moved that a letter of sympathy be addressed to Mr. Neilson Hancock in his recent sad bereavement. The motion was carried in silence, all members standing.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Raysmith, Frank Walker, L.R.C.P. (Edinburgh), 1928, L.R.C.S. (Edinburgh), 1928, L.R.F.P.S. (Glasgow), 1928, George Street, Brooklyn, New South Wales.

Peoples, Charles John, L.R.C.P. (Edinburgh), L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1927, 4, Cook Road, Centennial Park.

McCaffrey, Christian James, M.B., 1925 (Univ. Sydney), District Hospital, Newcastle.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Boag, James Fraser, M.B., Ch.M., 1925 (Univ. Sydney), 92, Birkeley Road, Manly.

Gourlay, Gwladys Dalrymple, M.B., Ch.M., 1925 (Univ. Sydney), 1, Highfield Road, Lindfield.

Graham, Colin Spencer, M.B., Ch.M., 1926 (Univ. Sydney), Lane Cove Road, Turramurra.

Hawker, John Kenrick Alva, M.B., 1928 (Univ. Sydney), Balmain District Hospital, Balmain.

Matthews, Henry Delahunt, M.B., 1908 (Univ. Sydney), Mallusk, Hilltop Crescent, Manly.

McManamey, John, M.B., B.S., 1928 (Univ. Sydney), 68, Upper Pitt Street, Milson's Point.

Windeyer, Brian Wellington, M.B., B.S., 1927 (Univ. Sydney), c.o. Australian Bank of Commerce, 62, Bishopsgate, London.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Kefford, Minnie Isobel Wilson, M.B., B.S., 1929 (Univ. Melbourne), 17, Kintore Street, Camberwell.

Macknight, Ella Annie Noble, M.B., B.S., 1928 (Univ. Melbourne), Melbourne Hospital.

Bottomley, Edward Eric, M.B., B.S., 1929 (Univ. Melbourne), 177, Cotham Road, Kew.

Farran-Ridge, Clive, M.B., 1915, Ch.M., 1917 (Univ. Sydney), Dip.P.M. (London), 1921, MacLeod.

Duncan, Lois Madge, M.B., B.S., 1929 (Univ. Sydney), Carlton, Victoria.

Altmann, Albert Adolph, M.B., B.S., 1929 (Univ. Melbourne), 10, Charnwood Road, St. Kilda.

Scott, Ian Mitchell King, M.B., B.S., 1929 (Univ. Melbourne), 249, Williams Road, South Yarra.

Tremaine-Park, Alexander, M.B., B.S., 1928 (Univ. Melbourne), Geelong.

Medical Societies.

BRISBANE HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE BRISBANE HOSPITAL CLINICAL SOCIETY was held on December 13, 1928, Dr. J. M. THOMPSON in the chair.

Duodenal Ulcer.

DR. E. S. MEYER presented a patient, an old age pensioner, who had been admitted to the hospital on October 23, 1928, with a history of having been quite well until eleven weeks prior to admission when he had had attacks of severe griping pain in the epigastrium. The pain had commenced in the right epigastric region and had travelled to both sides of the abdomen and into the inguinal region where there was a hernia. It was absent in the morning and on walking. It had not occurred every day, but had appeared after a fish meal and frequently in the evening. One week after the pain had appeared he had had an attack of vomiting about three hours after food. The pain had been relieved by vomiting and by taking milk. He had been constipated and at times his abdomen had been distended. There had been blood in the motions, but the patient had suffered from piles. He had been told at the Cunnamulla Hospital that there had been blood in his vomitus.

The onset had been sudden; he had had no previous indigestion and no jaundice. His appetite had been indifferent and he had lost 12·7 kilograms (two stone) in weight. There had been no urinary symptoms. Examination had revealed that the heart and lungs were normal. The abdomen was slightly tender and slightly rigid on the right side. The patient had been treated in the medical

ward for duodenal ulcer by Sippy's diet. A barium meal and X ray examination had been carried out. A residual duodenal fleck with pyloric spasm and dilatation of the antrum had been noted. There had been a slow passing of the fluid and an accompanying dilatation of the stomach. The diagnosis was duodenal ulcer.

Dr. Meyers had operated on the patient on November 22, 1928, after some septic teeth had been removed. A pararectal incision had been made and a hard mass had been found in the pyloric region about 2·5 centimetres in length. Dr. Meyers had been uncertain whether this mass was an ulcer or a neoplasm. He had performed a modification of the operation of partial exclusion; the distal end of the stomach had been closed and the proximal end joined to the jejunum. The anastomosis was anticolic. No enlarged glands had been found. After the operation the patient had been given meals according to the following plan: at 5.30 a.m. a cup of tea, at 8.0 a.m. a third of a litre of cornflour, at 11.0 a.m. a third of a litre of beef tea, at 12.30 p.m. a third of a litre of junket, at 5.0 p.m. a third of a litre of gruel and at 7.0 p.m. a third of a litre of beef tea. A cup of milk had been taken occasionally in the afternoon. On admission the patient's weight had been forty-seven kilograms (seven stone ten pounds) and at the time of the meeting it had increased to fifty-three kilograms (eight stone five pounds). Dr. Meyers remarked that it would be possible to implant radium into the distal end of the stomach.

Tumour of the Parotid.

Dr. E. S. Meyers presented a patient from whom he had excised a parotid tumour. A basal celled growth had also been removed by diathermy. The original wound for the removal of the tumour of the parotid gland had cicatrized well in two months and the patient's general condition had become moderately good. At the operation Dr. Meyers had had much difficulty with the auditory meatus. His patient had been fortunate in that he could hear on the affected side.

Hodgkin's Disease.

DR. ALAN LEE showed a patient for Dr. Eustace Russell. The patient had paralysis of the right vocal cord due to paralysis of the right recurrent laryngeal nerve. The only symptom had been the appearance of an enlarged gland in the occipital region seven weeks previously. After this his voice has become altered. No other enlarged glands had been noted. The diagnosis lay between Hodgkin's disease and Sternberg's disease. Dr. Lee raised the question whether Sternberg's disease was or was not a mediastinal form of Hodgkin's disease.

Polycystic Disease of the Kidney.

DR. C. M. LILLEY showed a patient for Dr. Eustace Russell. The patient's symptoms had been giddiness and dizziness for six weeks. Seventeen years previously the patient had had a fibroid growth removed from the abdomen. Four years previously she had been in hospital on account of giddiness and since that time she had had attacks of vomiting. There had been no œdema of the ankles, but the abdomen had been distended at times. There had been difficulty in commencing micturition and some nocturnal frequency. At times she had had pain on passing urine toward the end of the act. No blood had been seen in the urine. She had been constipated. During the previous twelve months she had had œdema and tenderness of the eyelids in the mornings. On examination a large tender mass had been felt in the abdomen, not attached to the liver. The area had been dull on percussion. The mass had not been adherent to the skin. The systolic blood pressure had been 175 millimetres of mercury. The urea content of the blood had been sixty milligrammes per hundred cubic centimetres. After the injection of indigo-carmine no dye had been excreted from either kidney in fifteen minutes. The diagnosis of polycystic disease of the kidney with dilatation of the renal pelvis was made from a pyelogram.

Traumatic Peritonitis.

DR. C. M. LILLEY presented a male patient who had suffered from an attack of abdominal pain ten days previously. The pain had become severe a week later. The patient had eaten fowl on the day before the pain had appeared.

The condition had been diagnosed as appendicitis and an operation had been performed. General peritonitis had been found; the appendix had been normal, but a fowl bone had been found skewered through the wall of the small intestine.

Renal Calculi.

Dr. Lilley also showed a male patient who had had a history of renal colic extending over several years. X ray examination had yielded a shadow at the lower end of the left ureter close to its entrance to the bladder. In a pyelogram four small and one large calculi were seen; there was a very small opening into the ureter. Dr. Lilley had come to the conclusion that the patient would be unable to pass the stones. He had to decide whether he would approach the calculi through the bladder or through the peritoneal cavity. He proposed to retain the kidney, although he realized that the patient might have more stones in the future and that it might be necessary to remove the kidney at a later date. While he was preparing the pyelogram the catheter had slipped past the stone. It was the first time that this had occurred to him.

Epithelioma.

DR. A. G. ANDERSON exhibited a patient, aged seventy-eight years, who had been admitted to the hospital on account of swellings on the right side of the neck. The patient had noticed the swellings for about seven or eight weeks. It was found that there was a hard, irregularly shaped solid mass of lymphatic glands fixed to the underlying tissues of the neck. The patient was a gardener, but there was no ætiological significance attaching to his occupation. Although no lesion was detected at first in the throat, Dr. Anderson had referred the patient to Dr. Culpin who had reported that there was a very extensive epitheliomatous growth at the base of the tongue, just above the epiglottis. A Wassermann test had been carried out and the serum had yielded a strong reaction. Dr. Anderson had concluded, however, that this finding had nothing to do with the growth. The condition was too far advanced for surgical treatment. It was determined to try the effect of radium.

Spinal Lesion.

DR. A. J. REYE presented a male patient who had been admitted from the out-patient department on account of various nervous symptoms. He had complained of fullness under the sternum, especially after food. He had had no pain, but merely discomfort. He had pain on the left side of the chest, slight headache over the left parietal and temporal areas and failing vision. He had found that he tired readily. A skiagram had revealed an outgrowth between the ribs and the aorta on the left side. There had been noted a difference of twenty millimetres between the systolic blood pressure measured in the right and the left arms. The patient had ataxia from which he had suffered for ten years. There had been no breathlessness, no cough and no sputum. His weight had not decreased. Examination of the abdomen had revealed nothing abnormal. The knee jerks were active, there was no Babinski's sign and no clonus. The pupils reacted to light and accommodation. The sensation had not been tested. No reaction had been obtained to the Wassermann test and Dr. Reye was satisfied that the ataxia was not of luetic origin. In spite of this he had treated the patient with antisyphilitic remedies and had prescribed reeducation exercises for the lower limbs. Dr. Reye raised the question of progressive cerebellar degeneration in the differential diagnosis.

Ulcerative Tumour of the Foot.

DR. C. WEEDON discussed a curious tumour on the dorsum of the foot of a male patient. Thirty years previously the

patient had had his right foot penetrated by the edge of a boot. The wound had not healed. Two years before admission a cyst had been removed from the right foot. Since that time a mass had been present and had been increasing in size and hardness. The tumour had ulcerated and pus had been discharged over the dorsum. The diagnosis of epithelioma had been made. A piece of the mass had been excised for microscopical examination. The pathologist had reported that there was hyperplasia and inflammatory reaction, but no trace of malignant changes. The patient's serum did not deflect complement in the Wassermann test. A skiagram had revealed a peculiar condition of the tibia, fibula and bones of the foot. At the lower end of the tibia there was some raising of the periosteum.

Congenital General Paralysis of the Insane.

DR. JOHN BOSTOCK exhibited two patients with congenital general paralysis of the insane. The first was a child with congenital syphilis. The child was an inmate of the deaf and dumb school. She had been bright until two years previously and then had gradually become dull and had moped. She had had increasing spasticity. Under protein shock treatment she had improved considerably.

The second patient was a boy, aged twenty-two years, who had had fits and had been regarded as a typical epileptic. His pupils were unequal; he could not remember events and had frequent headaches. He had said that his head felt strange. He was thinking of something else and forgot what he wanted to say. He had a coarse tremor of the tongue. He had exaggerated knee jerks, wrist and jaw reflexes. The Wassermann test had not been carried out. Dr. Bostock stated that the prognosis in these patients was worse than it was in adults suffering from acquired syphilis and ordinary general paralysis of the insane.

Obituary.

BENJAMIN ROBERT RICHARDS.

IN our issue of April 13, 1929, the announcement was published of the death of Benjamin Robert Richards on April 1.

Benjamin Robert Richards was born on January 11, 1887. His father was for many years schoolmaster at Box Hill, Victoria. He died in 1914. Benjamin Robert Richards was educated at the Box Hill Grammar School. He distinguished himself in sport. At the age of nineteen he entered the Medical School of the University of Melbourne. His course was a successful one. While he manifested much ability at his work, he also was in the front rank on the playing fields. He gained his "blue" for football and did well in other branches of sport. In 1910 he took his degrees in medicine and surgery. After graduation he served as a resident medical officer at Saint Vincent's Hospital for a year and then he entered practice at Box Hill. He soon became popular and his practice grew rapidly. He was fond of surgery and he seized every opportunity to render himself proficient. He was dexterous as an operator and his judgement was sound.

In 1916 he joined the Australian Army Medical Corps Reserve with the rank of honorary Captain.

He was a very popular man, jovial, kindly and generous. In his practice he worked hard and did not consider himself. He was a keen sportsman and was a particularly fine shot. He acted as a member of the Committee of the Melbourne Gun Club for a considerable time. He was also fond of fishing and of golf. He was the first president of the Easham Golf Club. In other sports he was also well known. He played cricket well and was a good swimmer. He married in 1913 and is survived by his widow and three children. The sympathy of the medical profession is extended to them.

DR. H. OSBURN COWAN writes:

To the sorrow of all who knew him, Dr. Benjamin R. Richards, of Box Hill, Victoria, a busy and popular medical man, passed away on April 1, 1929, at the early age of forty-two years.

Known to a wide circle of friends as Ben and to his patients as Dr. Ben, he radiated good nature, abounding energy and a keen interest in all forms of active life. While at the Melbourne University he was awarded his blue for football and is still remembered as one of the best when 'varsity football was at its best.

During his term as Resident at St. Vincent's Hospital he had the privilege of working under Dr. (now Sir Douglas) Shields and profited greatly from his close association with the dexterous and resourceful operative work of this distinguished surgeon. Especially did he recall the extreme respect for tissue which was such a marked feature of Dr. Shield's technique. Going into general work at Box Hill he soon acquired an extensive practice and availed himself of the opportunities it afforded for general and especially for emergency surgery.

One of the kindest of men, he gave his best most generously to his work and never failed to take into account the human equation. Over frequent periods he toiled incessantly day and night and in the end largely lost his capacity for sleep. Strong as he was, this gradually wore him down and he passed away many years before his time. The profession extends its sympathy to his widow and three children and assures them that Dr. Ben Richards has a warm place in many hearts, both within and without its ranks.

CHARLES AUGUSTUS EDWARDS.

WE regret to announce the death of Dr. Charles Augustus Edwards which occurred at Sydney on May 7, 1929.

Correspondence.

SUPPURATING HYDATID DISEASE OF THE LUNG CURED BY ARTIFICIAL PNEUMOTHORAX.

SIR: Dr. Cotter Harvey's case described under the above heading in your issue of the sixth of April is an excellent example of the classical method of treating abscessed cavities in any situation. At first sight this may not appear apparent. The causal agent need not concern us, but only the fact that an abscess was present in the lung. Before the operation the abscess was draining, but imperfectly so, through a bronchus which Dr. Harvey correctly compared to a drainage tube of rubber. Collapse of the lung caused the cavity to be squeezed almost dry of its contents. We have now an empty cavity efficiently drained by a rigid tube emptying into the antiputrefactive upper respiratory tract, an arrangement in accordance with the principle of the antiseptic treatment of wounds, abscessed or not.

Under these conditions we know that the need for continued formation of pus is at an end and no more pus will be secreted by the walls of the abscess. Cure by cicatrization and adhesion of the opposing walls must inevitably ensue, the end-result of an end-process. This Listerian conception of the pathology of abscess, acute or chronic, is seldom seen nowadays, in fact it is only demonstrated by the fortuitous provision, as in this case, of the conditions for carrying out what is known as the antiseptic principle. I congratulate Dr. Harvey on his choice of method, even if he has not appreciated its rationale.

Yours, etc.,

A. C. F. HALFORD.

Wickham House,
Brisbane,
April 6, 1929.

TUBERCULOSIS AND CANCER.

SIR: The concomitant variation which Dr. Cherry has so ably demonstrated from his study of stupendous statistics to exist between the incidence of tubercle and cancer, has

drawn me back in acute memory, some ten years to a time, when I was much intrigued in the connexion between these two diseases or between these two groups of diseases. I was profoundly interested in the recurrence after operation. I had travelled as far as considering the disease cancer as comprising several conditions, at times I even thought that cancer was a differing affair in each organ. It was often in my mind that the arcanum of cancer would not become manifest necessarily to the highly scientific seeker, but that a general observer might stumble on something, which might lay bare the secret. At the time of which I speak, there happened to come together in my observation several cases in which I had operated for mammary cancer and the patients had remained well for many years, twelve thirteen and nine, until there developed an enlargement of a gland at some distance from the site of operation. This was very disquieting for both patient and surgeon and provocative to the surgeon. It would have been very schismatic to put down the enlargement in the gland to a new cancerous growth, but if not, what had prevented the cancerous element from growing all the time. The non-growth was to me in the nature of an inhibition rather than the result of a combat; I knew that human cells could engage and eventually in some cases destroy cancer cells and that cancer cells, whatever their origin may be, could in most cases overcome all human cells, but this was agonistic and one or other must conquer finally. How then could the growth of the cancer element left over by operation have been rendered impossible so long. We can imagine the cancerous element just able to exist somewhere between the operation area and the cervical gland halfway up the neck and then carried into the structure of the gland, where later it was able to proliferate. What kept it from proliferating all those thirteen years in my patient? This gland was removed by myself and microscoped by Dr. Mollison. It was undoubtedly carcinomatous. The inhibition had lifted and other enlargements appeared in the neck and the pleura on the same side filled with fluid. The patient refused further treatment and died miserably some two months after. Such a terrific *dénouement* after such a period of apparent cure surely is best explained in its large area of action, by the removal of some restriction. Here is a portion of tissue the urgency of whose life history is to grow. It stultifies its life history by not growing. Is it not natural to look for some inhibition capable of acting over a large area? The cells of the body could not produce such an equilibrium lasting so many years; either cell wins or loses. If not to solid, we must look to liquid. Tissue fluid, the fluid which bathes the tissues normally, allowing for their tension, removing part at all events of their refuse, acting everywhere and altering in constitution perpetually, surely this tissue fluid is able by its nature to provide an inhibitory action over a large area. This will vary and may fail after a time or it may not furnish a sufficiency of inhibition at first.

If it can be imagined to be as full of inhibitory virtue as all this, why did it not prevent the original growth? It may be logically said that while the human body seems always to aim at resistance to obnoxious influences, such resistance does not appear until the obnoxious influence is at work some time. We know that some cancerous mammary growths do pause in their career for years and we also know that a severe shock, for example an operation for atrophic cancer in the very senile breast, will neutralize such inhibition and a cancerous debauch will ensue in the tissues over a very extensive area.

We know that the composition of the tissue fluids of the body is always altering. It is reasonable to surmise that some anticancerous elements reach the fluids from the body which resents so many things. If we admit this anti-element in the constantly altering fluid, we can admit its antiness sufficiently strong to nullify cancerous growth at times and in a fluid which is not constant, the content may be changed towards the trough or the summit of the antiwave with consequent allowance of the growth of the cancer cell or its nullification.

It is possible to stimulate cells, but their action must be more or less local. Fluids from their very nature must be credited with more pervasive possibilities. Could the tissue fluids be altered so as to make them more antagonistic to cancerous development?

Certain drugs certainly affect the composition of the tissue fluid in this way for a time, for example potassium iodide will diminish the size of a mammary cancer if given internally. But better I thought would be something organic if I could get it. Then I thought of what disease might be said to be antagonistic to cancer as evidenced by not occurring with it. Of course, my mind soon broke itself against disease caused by tubercle bacilli.

My friends Allen and Mollison added to my own experience, theirs concerning cancer and tubercle occurring at the same time in a human individual.

I knew from common observation that tuberculin manufactured in the body of the patient was a most pervasive addition to the tissue fluids. It alters every part of the person, his thermotaxic apparatus, his brain activities, his sexual outlook. Nothing could be more searching than this tuberculin in the avenues and alleys of the body. Now I did not care to say that this tuberculin (old) prepared outside the body could be anticancerous in its multiform activity, but I did say to myself that it would alter the composition of the tissue fluids in some way; some alterative to the fluid was indicated in my mind.

It was not very long before a case came to me that afforded a justifiable experiment, a lady from Western Australia upon whom I had operated for cancer of the breast many years before (Dr. Mollison always confirmed the diagnosis of these cases microscopically) came back with a large lump, which had recently formed at the apex of the axilla. There was some swelling of the arm. I advised further operation which was proceeded with, but I found the mass investing very closely the blood vessels and nerves and it seemed to me that nothing short of amputation would allow of suitable removal. This mutilation was refused and I was begged to try anything that I thought might help. I began with a diagnostic dose of old tuberculin which produced no reaction and then gradually increased the bi-weekly dose, till I reached heroic dosage, namely half a milligramme and even one milligramme. After some weeks these very big doses produced painful ulcers at the point of subcutaneous injection. The patient herself improved as the dosage increased, feeling much better and after a time my son and I allowed ourselves to think that the lump had decreased and in three weeks there was no doubt about it. After two months the lump disappeared, the swelling of the arm subsided and two years later the lady was well without any sign of cancer. Mr. Bull, biochemist who prepared the tuberculin for me, warned me of the toxic nature of my dosage. I may add that I thought after my second operation she could not live more than two or three months, so rapid was her decline.

The next case in which tuberculin was used was that of a little woman, weighing about six stone, whose breast had been removed, but not by myself, some eight months before. She was an embodiment of what our fathers would have called cancerous cachexia. She had some small nodules in the vicinity of the operation scar, which could very reasonably be called cancerous. She had much pain in her back which was very bent. She could hardly walk and her general condition was very bad. She gave no reaction to the diagnostic injection of tuberculin. Gradually increasing doses were given and after two months' rest and treatment in my private hospital she was able to return to her husband's farm and do her ordinary housework apparently cured. The nodules had disappeared. The patient had gained much flesh and regained as good health as she had ever enjoyed. She died of influenza over a year after this.

A third case in which another surgeon had operated, asked me to treat her when she was advanced in a recurrent carcinomatous outburst. She improved with the injections for some weeks, but it was asking more than any treatment could give, as her whole torso was studded with nodules of carcinoma. Even then we were hopeful for a time.

In each of these cases the patient after a few injections expressed herself as feeling much better.

It is not easy to get suitable cases, when one wants them for experiment and my own health failing I was constrained to abandon this treatment which seemed as if it might add much to our ability of dealing with carcinoma of the breast. It has interested me exceedingly to find that the giving of tuberculin is a recognized treatment at the Johns Hopkins Hospital, Baltimore, in discriminated cases of recurrent carcinoma. How the discrimination is arrived at I have not yet been able to learn.

Yours, etc.,

FRED D. BIRD.

Melbourne,
April 11, 1929.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE Executive Committee of the third session of the Australasian Medical Congress (British Medical Association) desires to call the attention of the members of the several Branches to the following matters.

Section of Preventive Medicine and Tropical Hygiene.

In the list of sections published in our issue of May 11 the Section of Preventive Medicine and Tropical Hygiene was inadvertently omitted. The office-bearers are as follows:

President, Dr. R. C. Everitt Atkinson, Perth, Western Australia. *Vice-Presidents*, Dr. R. Dick, Sydney, New South Wales; Professor C. E. Hercus, Dunedin, New Zealand; Dr. F. S. Hone,, Adelaide, South Australia; Dr. C. N. Atkins, Hobart, Tasmania; Dr. J. Dale, Melbourne, Victoria. *Secretary*, Dr. E. S. Morris, Department of Public Health, Sydney.

Programme of Meetings of Sections.

Notice of papers and exhibits to be presented at the meetings of the sections must be received not less than sixty days before the inaugural meeting of Congress and a complete typewritten copy of the papers must be in the hands of the honorary secretary of the section not less than thirty days before the inaugural meeting. Members who propose to read papers at the third session are therefore required to give notice either to the joint Honorary Secretaries of the session, Dr. T. W. Lipscomb and Dr. A. A. Palmer, or to the secretaries of the sections not later than July 4, 1929, and to place their papers in the hands of the secretaries of sections not later than August 3, 1929. The work of the secretaries of sections will be facilitated by a strict observance of this rule.

Congress Dinner.

The Congress dinner will be held on Wednesday, September 4, 1929, in the dining hall of David Jones, Limited, Elizabeth Street, Sydney. Members will be seated in groups corresponding to the sections of Congress. Members who intend to be present at the dinner, are requested to forward to the joint Honorary Secretaries the sum of one guinea, with exchange added, except when the cheques are drawn on a Sydney bank, and to indicate the section with which they desire to be associated. Early notification of the intention to be present at the dinner will facilitate the work of the committee in charge of the arrangements.

Travelling Arrangements.

The Railway Commissioners in the several States of Australia have announced that the following concessions will be granted to members of Congress travelling to Sydney and returning to their homes. In accordance with the usual practice members travelling from other States will be issued return tickets at single fares *plus* one-third, provided that not less than six members attend from that State. The concession will apply to the wife of a member if she accompanies her husband. The tickets will be issued so as to enable members to arrive at their destination not more than three days prior to the commencement of the session of Congress and to begin the return journey within one month from the date of the inaugural meeting. The journey cannot be broken, except for two days when passing through a capital city. For this purpose Kalgoorlie and Goulburn are treated as capital cities.

Members and their wives residing in the country in New South Wales will be allowed return tickets at single fares and one-third. The journey cannot be broken. Coupons for return tickets must be presented for redating within one month from the date of the issue of the tickets.

The New Zealand Government Railways Department has advised that members and their wives and children, if accompanying them, will be issued first class tickets at holiday excursion fares. The tickets will be issued for use on the outward journey within fourteen days of the commencement of the session. In view of the distance from Sydney the Department has consented to extend the availability of the tickets from one to two months.

Sir Ewen Maclean.

Sir Ewen Maclean, the President of the British Medical Association, who will attend Congress as official delegate of the Association, proposes to travel to Australia *viâ* San Francisco and New Zealand. He will arrive in Sydney during the last week in August.

Ladies' Committee.

At a meeting held at 30 to 34, Elizabeth Street, Sydney, on May 8, 1929, a ladies' committee of Congress was formed, its function being to advise and assist the Executive Committee of Congress in the entertainment of the wives and lady relatives and friends of visiting members. Mrs. G. H. Abbott was appointed President and Mrs. C. Bickerton Blackburn Honorary Secretary of the committee. An executive committee was elected, composed of the following ladies: Mrs. H. C. Adams, Mrs. W. F. Burfitt, Mrs. Gordon Craig, Mrs. E. W. Fairfax, Dr. Marie Hamilton, Mrs. T. W. Lipscomb, Mrs. H. J. Marks, Mrs. J. Morton, Mrs. Clarence Read, Mrs. C. Nigel Smith, Mrs. R. B. Wade, Mrs. J. C. Windeyer and Mrs. Ralph Worrall. A general committee was appointed consisting of the following ladies: Mrs. W. G. Armstrong, Mrs. J. E. V. Barling, Mrs. Fourness Barrington, Mrs. C. G. Berge, Mrs. H. H. Bullmore, Mrs. A. W. Campbell, Mrs. Victor Conrick, Mrs. F. Brown Craig, Mrs. St. John Dansey, Mrs. Andrew Davidson, Mrs. J. A. Dick, Mrs. Robert Dick, Mrs. H. C. E. Donovan, Mrs. Leslie Dunlop, Mrs. B. T. Edye, Mrs. J. Foreman, Mrs. A. J. Gibson, Mrs. Sinclair Gillies, Mrs. D. J. Glissan, Mrs. R. S. Godsall, Mrs. R. A. R. Green, Dr. Lucy Gullett, Mrs. Garnet Halloran, Mrs. Clifford Henry, Mrs. Holmes à Court, Miss Hood, Lady Howse, Mrs. E. M. Humphery, Mrs. J. G. Hunter, Mrs. W. K. Inglis, Mrs. E. J. Jenkins, Mrs. P. Sydney Jones, Mrs. H. W. Kendall, Mrs. C. H. E. Lawes, Mrs. M. C. Lidwill, Mrs. C. G. McDonald, Mrs. E. H. Molesworth, Mrs. E. S. Morris, Mrs. Earle Page, Mrs. H. R. G. Poate, Mrs. J. S. Purdy, Mrs. Cecil Purser, Mrs. H. R. Sear, Mrs. R. Scot Skirving, Mrs. A. M. Stanton, Mrs. E. H. M. Stephen, Mrs. B. P. Anderson-Stuart, Mrs. R. J. Taylor, Mrs. W. Vickers, Mrs. Granville Waddy, Mrs. A. P. Wall, Mrs. J. F. Watson, Dr. Mary Burfitt Williams.

The committee requests all medical practitioners who propose to become members of Congress, to intimate on the forms of application to be sent to the local secretaries whether they will be accompanied by ladies or not, in order that satisfactory arrangements may be made. These forms should be sent in as early as possible.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

The undermentioned have been registered under the provisions of *The Medical Act*, 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Dawson, Henry Gray Woodhouse, M.B., Ch.B., 1909 (Univ. Sheffield), 75, Darlinghurst Road, Darlinghurst.

Frazer, Edward Haslett, M.B., B.Ch., 1920 (Univ. Dublin), 231, Macquarie Street, Sydney.

Freedman, John Nathaniel, M.B., B.S., 1926 (Univ. Melbourne), Kurri Kurri.

Books Received.

DISEASES OF CHILDREN, Second Edition, Edited by Hugh Thursfield, D.M. (Oxon.), M.A., F.R.C.P., and Donald Paterson, M.B. (Edin.), M.R.C.P.; 1929. London: Edward Arnold and Company; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 1116, with illustrations. Price: 54s. net.

DISEASES OF THE EAR, NOSE AND THROAT: MEDICAL AND SURGICAL, by Wendell Christopher Phillips, M.D.; Seventh Revised and Enlarged Edition; 1928. Philadelphia: F. A. Davis Company; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 942, with illustrations. Price: 45s. net.

RADIUM TREATMENT OF CANCER, by Stanford Cade, F.R.C.S. (England); 1929. London: J. and A. Churchill; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 166, with illustrations. Price: 18s. net.

MANSON'S TROPICAL DISEASES: A MANUAL OF THE DISEASES OF WARM CLIMATES, Edited by Philip H. Manson-Bahr, D.S.O., M.A., M.D., D.T.M. and H. (Cantab.), F.R.C.P. (London); Ninth Edition, Revised; 1929. London: Cassell and Company, Limited; Sydney: Angus and Robertson, Limited. Demy 8vo., pp. 941, with illustrations. Price: 38s. net.

ANNALS OF ROENTGENOLOGY: A SERIES OF MONOGRAPHIC ATLASES, Edited by James T. Case, M.D.; Volume VIII: The Vertebræ; 1929. New York: Paul B. Hoeber, Incorporated. Demy 4to., pp. 282. Price: $10.00 net.

Diary for the Month.

MAY 21.—Tasmanian Branch, B.M.A.: Council.
MAY 21.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 22.—Victorian Branch, B.M.A.: Council.
MAY 24.—Queensland Branch, B.M.A.: Council.
MAY 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 28.—Illawarra Suburbs Medical Association, New South Wales.
MAY 30.—New South Wales Branch, B.M.A.: Branch.
MAY 30.—South Australian Branch, B.M.A.: Branch.

Medical Appointments.

Dr. Charles Firth Pettinger (B.M.A.) has been appointed Government Medical Officer at Wallsend, New South Wales.

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Dr. Geoffrey Wilson Morey has been appointed Honorary Aural Surgeon (Visiting) at the "Mareeba" Babies' Hospital, South Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.

COMMONWEALTH REPATRIATION COMMISSION: Medical Officer.

ROYAL PRINCE ALFRED HOSPITAL, SYDNEY: Honorary Medical Officer.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY: Senior Resident Medical Officer, Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Stannary Hills Hospital. Boonah District Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

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